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Experimental and Comparative Analyses of Maternal Age and Senescence

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Contents

Abstract

Senescence is often described as an age-related physiological deterioration accompanied with declining fertility and increasing mortality, and it is believed to be the result of declining forces of natural selection. A manifestation of senescence that has attracted much recent interest is the detrimental effect of increasing maternal age acting on offspring traits. However, uncertainty arises when attempting to describe the prevalence and ubiquity of this third form of ageing and the evolutionary causes for diversity in ageing trajectories. Here I address the following questions: (1) *How are maternal age effects distributed across taxa?* And (2) *Can an evolutionary perspective help us to understand the observed diversity in maternal age effects and demographic senescence?* I addressed these through (i) a cross-fostering ageing experiment using a laboratory population of burying beetle, *Nicrophorus vespilloides* to decouple the separate effects of increasing pre- and postnatal maternal age, whilst accounting for the potential bias of selective disappearance. I found no evidence for maternal age effects or effects deriving from selective disappearance. These results suggest that current theory may be insufficient to account for the true diversity in ageing patterns. (ii) A meta-analytical review of maternal effect senescence to investigate the prevalence and diversity of maternal effect ageing patterns and the performance of an evolutionary model to predict observed patterns. We found taxa-wide evidence for maternal age effects on offspring survival. However the direction of these effects was based on phylogenetic constraints with laboratory and natural-mammal species showing a decline, but natural-bird species

showing an ambiguous effect of maternal age. The evolutionary model was shown to improve in performance compared to evolution-agnostic demographic models when describing maternal effect ageing in natural populations. This result suggests an evolutionary cause to maternal effect senescence. (iii) Lastly, I performed a comparative analysis of vital rate selection across the tree of life. Using extensive existing databases of life history data coupled with predictions from two evolutionary theories, I derived correlations between predicted and observed vital rates across multiple animal species. I found that whilst natural selection had weak predictive power when describing patterns of mortality, age-specific fertility patterns showed extensive departures from evolutionary predictions. Additionally, I found that several biological processes were readily contributing to non-conformance of Hamilton-like ageing. Taken together, we provide convincing evidence to suggest that both natural selection and biological processes have helped shape the vast diversity of observed ageing rates that exist across the tree of life.

Lay Summary

Ageing is often accompanied with a decrease in fertility and survival, and is believed to be the result of declining forces of natural selection. One form of ageing that has attracted much recent interest is the harmful effect of increasing maternal age acting on offspring traits. However, there is uncertainty when attempting to describe the distribution of this form of ageing and the evolutionary causes for diversity in ageing patterns that exist across the tree of life. Here I address the following questions: (1) *How are maternal age effects distributed across animal species?* And (2) *Can a knowledge of evolution help us to understand the observed diversity in maternal age effects and traits related to ageing?* I addressed these through (i) an ageing experiment using a laboratory population of burying beetle, *Nicrophorus vespilloides* to separate the effects of increasing age of maternal egg-producer and carer, whilst also accounting for the possibility that older females in the population were of higher overall quality than younger females. I found no evidence for maternal age effects or that the quality of older females were biasing our results. These results suggest that current theory may be insufficient to describe all forms of ageing. (ii) A meta-analytical review to investigate the distribution and diversity of maternal effect ageing patterns and the performance of an evolutionary model to predict the types of ageing that we observe. We found widespread evidence to suggest that maternal age was having a significant effect on offspring survival. However the direction of these effects was based on phylogenetic constraints with laboratory and natural-mammal species showing a decline, but natural-

bird species showing an ambiguous effect of maternal age. The evolutionary model was shown to improve in performance when describing maternal effect ageing in natural populations. This result suggests an evolutionary cause to maternal effect ageing. (iii) Lastly, I performed a comparative analysis comparing fertility and mortality against levels of natural selection across the tree of life. Using extensive existing databases of life history data coupled with predictions from two evolutionary theories, I analysed correlations between predicted and observed values for mortality and fertility across multiple animal species. I found that whilst natural selection had weak predictive power when describing patterns of mortality, natural selection had even weaker power when describing patterns of age-specific fertility. Additionally, I found that several biological processes were readily contributing to the low predictive power of evolutionary theory. Taken together, we provide convincing evidence to suggest that both natural selection and biological processes have helped shape the vast diversity of observed ageing rates that exist across the tree of life.

Declaration

The work described in this thesis has been carried out by myself with guidance from my supervisor, unless otherwise stated and detailed below. The thesis is of my own composition and has not been submitted for any other degree or professional qualification.

For Chapters 2-4: E. Ivimey-Cook and Jacob Moorad conceived the ideas, designed the methodology, analysed the data and wrote the manuscripts; EIC collected the data. Both authors contributed critically to drafts and gave final approval for inclusion in this thesis.

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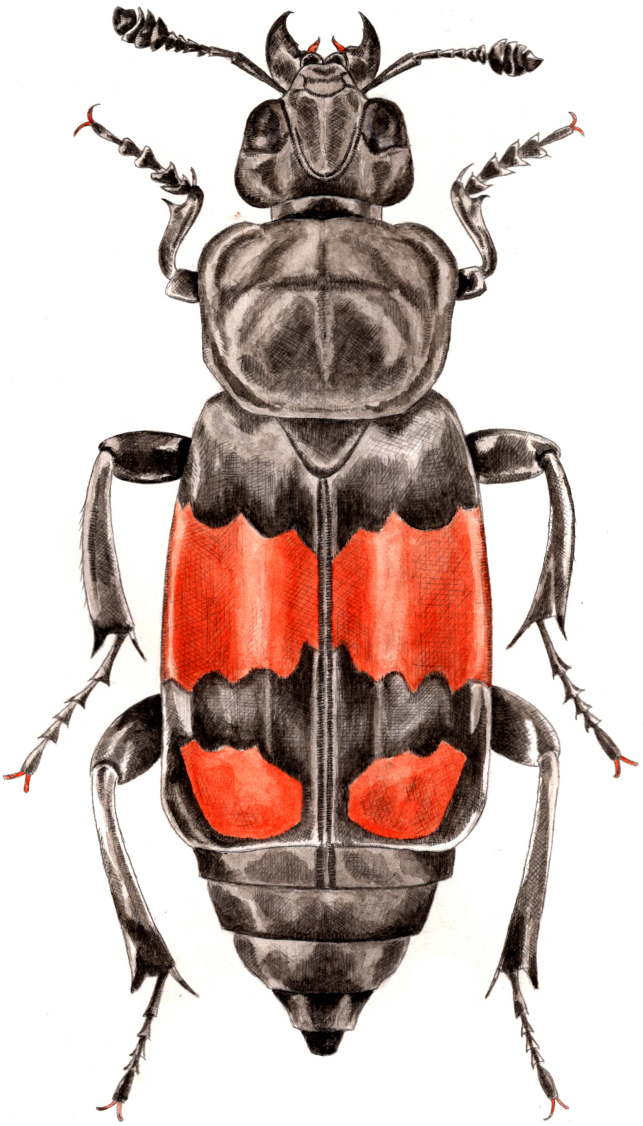

Special thanks go to Apithanny Bourne for illustrating this lovely female *Nicrophorus vespilloides* for my thesis.

Chapter 1: General Introduction

1.1 Senescence

Senescence is almost universally regarded as the progressive decline in physiological function and fitness with increasing organismal age (Hughes *et al.* 2002; Williams & Day 2003). Of particular importance are declines in survival (actuarial senescence) (Finch *et al.* 1990; Promislow & Harvey 1991; Partridge & Barton 1993; Jones *et al.* 2014), fertility (reproductive senescence) (Partridge & Barton 1993; Jones *et al.* 2014; Lemaître & Gaillard 2017) and, more recently, changes in neonatal survival as a result of increasing maternal age (maternal effect senescence, see Moorad & Nussey 2016). To date, senescent declines in survival and fertility have been widely documented in natural populations of wild vertebrate species and in captive populations of laboratory invertebrates (Rose 1984; Kenyon *et al.* 1993; Gaillard *et al.* 1994; Bonduriansky *et al.* 2008; Descamps *et al.* 2008; Jones *et al.* 2008, 2014; Nussey *et al.* 2008a, 2011; Bouwhuis *et al.* 2009; Galliot 2012; Waugh *et al.* 2015). However, it was only until the recent surge of longitudinal data was it concluded that actuarial and reproductive senescence were in fact common occurrences in wild vertebrates (Clutton-Brock & Sheldon 2010; Nussey *et al.* 2013). This is in stark contrast to the widely-held belief that species in natural environments would not survive to the ages where senescence actually begins to manifest (Medawar 1952).

1.1.1 Maternal effect senescence

Of particular relevance to the research conducted in this thesis is the transgenerational effects of maternal age on offspring traits such as size, growth and survival (Fox 1993; Fox & Dingle 1994; Mousseau & Fox 1998; Hercus & Hoffmann

2000; Kern *et al.* 2001; Priest *et al.* 2002; Fox *et al.* 2003; Descamps *et al.* 2008; Nussey *et al.* 2009; Beamonte-Barrientos *et al.* 2010; Froy *et al.* 2013). Whilst it was understood that maternal effects constituted a major part in the evolution of ageing, it wasn't until the development of more evolutionary theory did we begin to fully understand the profound impacts of maternal effect ageing on traits vital to fitness. Specifically, it was recent evolutionary theory by Moorad and Nussey (2016) that aimed to characterise maternal effect ageing acting particularly on neonatal survival, as distinct from fertility senescence. In particular, they provided a theoretical model which incorporated indirect genetic effects (IGEs) from social interactions into existing evolutionary theories of ageing (Hamilton 1966; Moore *et al.* 1997; Wolf *et al.* 1998, 1999; McGlothlin *et al.* 2010). From a quantitative genetics perspective, IGEs create an environment that can respond to selection (Hadfield *et al.* 2011) and, as such, cause the environment-term in classical quantitative genetics models (*Phenotype* = *Genotype* + *Environment*) to become partly heritable (Bijma 2014). As social interactions are likely to be commonplace in most natural, age-structured populations, we should then expect that any expressed variation in genes or environmental quality provided by other conspecifics to readily alter the measured phenotype of the focal individual (Wolf *et al.* 1998; Moorad & Nussey 2016). By integrating this quantitative genetic concept into the Evolutionary Theory of Ageing, Moorad and Nussey subsequently provided evidence that traits under the influence of these IGEs (such as maternal effects) are a distinct form of ageing. Additionally, they provided evidence of qualitative differences that can evolve between maternal effect ageing and other forms of trait senescence, including early-life improvements in neonatal survival followed by faster-than-Gompertz declines in late-life with accompanying age-related increases in genetic variance. However, whilst the diversity

and distribution of more traditional manifestations of senescence have been extensively investigated (see Jones *et al.* 2008, 2014; Lemaître & Gaillard 2017), the prevalence of these maternal age effects, whilst garnering increased attention, remain poorly described and understood (Bloch Qazi *et al.* 2017).

1.1.2 Diversity in ageing patterns

The observed diversity in ageing trajectories pertaining to actuarial and reproductive senescence has been an area of exploration for bio-gerontologists and evolutionary biologists for decades. To date, there has been a multitude of expansive comparative studies investigating age-related variation in trait patterns between and within taxa of animals (Promislow & Harvey 1991; Ricklefs 1998; Ricklefs & Scheuerlein 2001; Ricklefs *et al.* 2003; Jones *et al.* 2008; Péron *et al.* 2010; Lemaître *et al.* 2013; Nussey *et al.* 2013; Lemaître & Gaillard 2017) and plants (Jones *et al.* 2014; Salguero-Gomez *et al.* 2016b). Originally, past research focused on identifying the observed variance in actuarial senescence (Finch *et al.* 1990; Promislow & Harvey 1991; Ricklefs & Scheuerlein 2001). However, other components of senescence have since been considered and comparatively reviewed, including fertility (Ricklefs *et al.* 2003; Lemaître & Gaillard 2017), body mass (Nussey *et al.* 2011), foraging behaviour (Froy *et al.* 2018) and declining neonatal survival through maternal effect ageing (Ivimey-Cook & Moorad 2018b). Thus far, comparative work has identified intriguing differences in ageing rates between taxa, namely birds and mammals, with the oft-quoted observation that birds show much slower senescent declines in survival compared to mammals of analogous size (Holmes & Austad 1995). Recent comparative analysis by Jones et al (2008)

reinforced this disparity, as they compared rates of both actuarial and reproductive senescence (combined as a single metric: Individual Fitness Component, IFC) in 20 populations of vertebrates from the Mammalian and Aves taxa. They concluded that the faster pace of life exhibited by mammal species (referring here to short lifespan and generation time, high reproductive and mortality rate, see Jones *et al.* 2008 and Tidiere *et al.* 2016) lead to faster rates of IFC decline.

1.1.3 Captive vs. natural populations

Whilst the observable differences in rates of senescence between and within taxa may represent differences in an organism's natural life history, environmental variation between captive and natural populations can also contribute to variation in ageing rates (Ricklefs & Scheuerlein 2001). In particular, captive or laboratory populations experience a reduction in environmental stress through a constant, *ad libitum* supply of food, a reduction in predation pressure and readily available veterinary care. As a result of this decrease in environmental variation, captive individuals (such as those in zoo enclosures) tend to experience a slower decline in survival (often measured as the slope of a Gompertz model of age-specific mortality) compared to wild counterparts (Lemaître *et al.* 2013), albeit this is largely influenced by a species' pace of life. In fact, the majority of evolutionary research into theories of ageing and lifespan revolve around laboratory studies conducted using three primary model organisms, namely *Drosophila melanogaster* (Rose & Charlesworth 1980; Rose 1984; Hughes & Charlesworth 1994), *Caenorhabditis elegans* (Kenyon *et al.* 1993; Guarente & Kenyon 2000; Kenyon 2010) and mice (Peto *et al.* 1975; Miller *et al.* 2002; Sedelnikova *et al.*

2004). This is potentially problematic if one considers that the lack of variation in the environment may be leading to qualitatively different patterns of ageing than those occurring in nature (Williams *et al.* 2006). As a result, some studies use genetically identical populations of species in order to tease apart genetic and environmental interactions that may be complicating the interpretations of ageing, however, relatively few have used this approach thus far (Kawasaki *et al.* 2008).

1.2 Evolutionary theories of ageing

In comparison to the number of biological or physiological theories of ageing, which range from the age-related increase in damage-inducing factors to the progressive loss of irreplaceable proteins and which number in the hundreds (Medvedev 1990), relatively few evolutionary theories of ageing have been suggested. The first discussions regarding the evolution of ageing began in the 19th century by August Weismann. His theory of "Programmed Death" proposed that ageing was an adaptation where older individuals were eliminated from a population in order to free up resources for younger generations (Weismann 1882). However, his theory has largely been dismissed and the concept of an "ageing gene" which promoted the programmable death has been questioned (Kirkwood & Cremer 1982). Subsequent evolutionary theories have focused on ageing either as an evolved by-product of adaptation (e.g. Antagonistic Pleiotropy, Williams 1957) or as a maladaptive response to weakening levels of natural selection (e.g. Mutation accumulation, Medawar 1952).

1.2.1 Mutation accumulation

One of the most well-known and studied theories of ageing is the Mutation Accumulation hypothesis (MA) proposed by Peter Medawar in 1952. This evolutionary theory considered ageing as purely a by-product of weakening forces of natural selection acting on the genome of an individual. When selection is strongest in early-life, detrimental mutations would be severely selected against and eliminated as a result of their large, negative effects on an individual's fitness. However, as selection declines it becomes less effective at eliminating these mutations and they begin to accumulate. This increase in late-acting deleterious mutations therefore leads to an increase in risk of intrinsic mortality. There is however mixed evidence in support of this theory (Rose & Charlesworth 1980; Mueller 1987; Hughes & Charlesworth 1994; Promislow *et al.* 1996; Pletcher *et al.* 1998; Moorad & Walling 2017; Flatt & Partridge 2018).

1.2.2 Antagonistic pleiotropy

A second classical evolutionary theory of ageing is the Antagonistic Pleiotropy hypothesis (A.P.), suggested by George C. Williams in 1957. This theory suggests that alleles with detrimental effects in late life but beneficial effects in early life are selected for as a result of stronger selection acting on younger age classes. This can be manifested in both phenotypic and genetic correlations between early- and late-life traits such as fertility and survival. Empirical support for this theory comes from various work performed with laboratory populations of invertebrates (Rose & Charlesworth 1980, 1981a, b; Walker *et al.* 2000; Marden *et al.* 2003) and wild vertebrates (Charmantier *et al.* 2006; Nussey *et al.* 2006, 2008b; Lemaître *et al.* 2015). A similar life history theory, which is often seen as the mechanistic or optimisation form of Antagonistic Pleiotropy

is the Disposable Soma theory (D.S., Kirkwood 1977). This theory suggests that natural selection will adjust limited energy resources into two distinct processes, somatic maintenance and reproduction. In this way, senescence becomes inevitable as natural selection favours investment into early reproduction over long-term somatic maintenance. As A.P. and .D.S represent similar evolutionary models of ageing, support for these theories are commonly associated (See Table 1 from Nussey *et al.* 2013).

1.2.3 The force of natural selection

Unlike previous evolutionary theories of ageing, William Hamilton's 1966 classical publication "The Moulding of Senescence by Natural Selection" contained the first mathematical, rather than verbal (e.g. Haldane 1943, Williams 1957), descriptions of how natural selection changes with age. Specifically, he proved mathematically that the force of natural selection acting on age-specific survival and fertility was non-increasing and declined with age. In particular, that selection acting on survival began to decline shortly after the onset of reproduction, but for fertility, selection is maximised at birth and declines with advancing age (Figure 1.1)

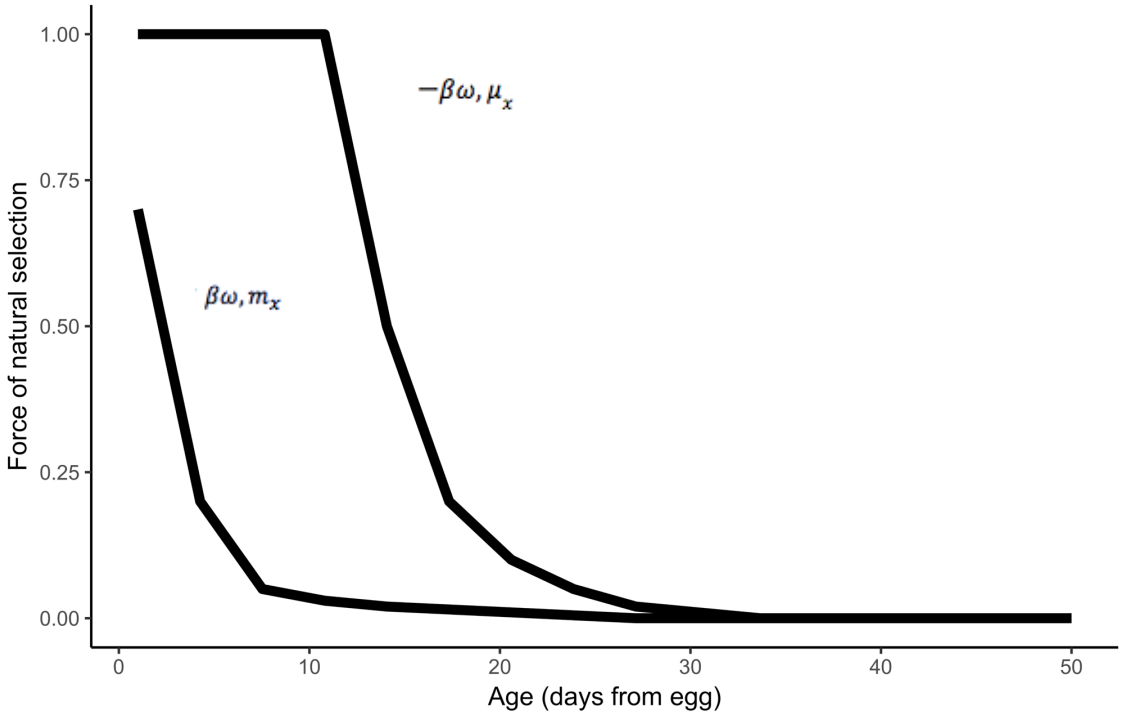

Fig 1.1: The strength of natural selection acting on age-specific fertility $\beta\omega, m_x$ and against mortality $-\beta\omega, \mu_x$ (adapted from Figure 1 in Rose *et al.* 2007, which represented fertility and mortality data from a popluation 1111 female Drosophila used in a study by Rauser *et al.* 2006).

Figure 1.1 shows explicit predictions made by Hamilton regarding the force of natural selection acting on vital rates. For age-specific survival, before the onset of sexual maturity selection acting on survival is always equal to one, representing an individual's future potential reproductive fitness. By the end of sexual reproduction selection is equal to zero, representing the lack of an individual's future fitness prospects.

1.3 Life history theory

Whilst evolutionary predictions made by Hamilton imply that traits such as survival and fertility should decline over the course of an adult's lifespan, several large-scale comparative reviews (Promislow 1991; Gaillard *et al.* 1994; Ricklefs & Scheuerlein 2001; Ricklefs *et al.* 2003; Jones *et al.* 2008; Péron *et al.* 2010; Nussey *et al.* 2013; Jones *et al.* 2014; Lemaître & Gaillard 2017) have provided evidence for a large diversity in ageing trajectories that appear to violate these predictions across multiple taxa, including increasing, decreasing, hump- and u-shaped patterns to age-specific fertility and survival. Apparent increased performance with age has been attributed to several life history processes that contribute to departures from predicted senescence patterns. Here I discuss the two main processes that are relevant to this thesis: selective disappearance or the selective mortality of poor quality individuals, and an increase in age-related reproductive effort. Other contributing factors are discussed in detail in Chapter 4. In truth, these life history models can all contribute simultaneously to departures from "Hamilton-like" ageing, however I will discuss each individual process in turn.

1.3.1 Selective disappearance

Often, the individuals that survive to the oldest ages are of higher general quality than those who have not survived. In wild vertebrates, this is frequently observed with older individuals having greater reproductive performance and body mass (Bérubé *et al.* 1999; Cam *et al.* 2002; Weladji *et al.* 2006). This "heterogeneity" in phenotypic traits and subsequent demographic rates presents a major challenge to ageing research, particularly if "frailer" individuals in a population die young, which leaves a non-random

high-quality subset of older individuals in the population. If left unaccounted for, this selective mortality can lead to underestimated rates of senescence or false inferences of age-related improvement (Hayward *et al.* 2013). Evidence of selective disappearance affecting phenotypic trait means has been found in body mass data of three ungulate species (Nussey *et al.* 2011), ageing trajectories of body mass in grey mouse lemurs (Hämäläinen *et al.* 2014), survival/breeding rates in a species of kittiwake (Cam *et al.* 2002) and, more recently, strong evidence of condition-dependent mortality in African killifish (Vrtílek *et al.* 2018). The effects of selective disappearance can be statistically corrected for (van de Pol & Verhulst 2006; van de Pol & Wright 2009; Nussey *et al.* 2011) by fitting longevity as a covariate. However, this technique has only recently been applied to account for selective disappearance in maternal effect ageing research (See Hayward *et al.* 2013; Berger *et al.* 2015; Fay *et al.* 2016b; Ivimey-Cook & Moorad 2018a).

1.3.2 Reproductive effort

As an individual ages, the potential for future reproduction decreases. In response, as the age-specific expectation of contributing progeny to the population decreases, an individual's reproductive value declines (calculated from cumulative survival l_x and fecundity m_x, $v_x = \sum_{t=x}^{\omega} \frac{l_t}{l_x} m_t$, Fisher 1930; Pianka & Parker 1975; Barton & Etheridge 2011). Consequently, life history theory predicts that the allocation of available resource and subsequent effort to reproductive processes should increase, known as the reproductive effort model (Williams 1966b; Charlesworth & Leon 1976). An organism will therefore aim to maximise lifetime reproductive fitness by balancing investment into both current and potential future breeding opportunities. However, If no future

opportunities exist, an individual should therefore invest all remaining resources into one final reproductive attempt, a process known as Terminal Investment (Clutton-Brock 1984). This increase in reproductive effort may lead to the characteristic quadratic form to ageing trajectories typical of many mammalian and bird species, with an early/mid-life increase followed by a senescent decline in late-life. Support for increased reproductive effort with age is mixed, with studies showing both age-related increases (Pugesek 1981; Poizat *et al.* 1999; Kight *et al.* 2000; Ericsson *et al.* 2001; Velando *et al.* 2006; Creighton *et al.* 2009; Paterson *et al.* 2016; Hargrove *et al.* 2018) and senescent declines (Dugdale *et al.* 2011; Martin & Festa-Bianchet 2011; Conover 2013; Kuczynski *et al.* 2015) in reproductive success with age.

1.4 Cross-fostering experiments

The basic rationale behind the use of cross-fostering techniques in experimental research is to separate the interacting effects of genes and environment which can potentially influence phenotypic traits (Kruuk & Hadfield 2007; Winney *et al.* 2015). In particular, this methodology has been successfully employed in a multitude of studies of both wild and laboratory populations in order to transfer offspring from their natal habitat to a new environment with novel social partners. Often this results in an improved understanding of phenotypic variation and response to selection acting on a population. There are potentially a number of troublesome problems that can arise from this technique, such as the disruption of parent-offspring communication (See Table 1 from Winney *et al.* 2015), but its use in decoupling the individual effects of pre- and postnatal maternal age emphasise the valuable benefits of this experimental approach.

Other, non-experimental techniques for disentangling genetic and environmental effects include the use of the mixed effects models also known as the animal model (See Falconer & Mackay 1996). This quantitative genetics technique has been widely used to investigate sources of variation in life history and morphological traits (Kruuk *et al.* 2000, 2002; Charmantier *et al.* 2004, 2006).

1.4.1 The use of cross-fostering to study maternal effects

In particular, cross-fostering allows us to disentangle the individual effects of increasing pre- and postnatal maternal care, as, often these two traits can have differing contributions to offspring performance (Beamonte-Barrientos *et al.* 2010; Lemaître & Gaillard 2017). Used in this way, cross-fostering allows for independent testing of age-related changes in egg quality (prenatal) and rearing capacity (postnatal) and can reveal potential differences in ageing trajectories for each trait (Lock *et al.* 2007; Beamonte-Barrientos *et al.* 2010). Cross-fostering has been used to investigate contributions of pre- and postnatal maternal effects manifested on offspring traits on several occasions, particularly in wild bird species (Bolton 1991; Bize *et al.* 2002; van de Pol *et al.* 2006) and in laboratory invertebrates, particularly from the *Nicrophorus* taxa (Lock *et al.* 2007; Trumbo 2009; Ivimey-Cook & Moorad 2018a).

1.5 The burying beetle, *Nicrophorus vespilloides*

Beetles of the genus *Nicrophorus* are typically characterised by their elaborate and facultative levels of bi-parental care. Of particular relevance to this thesis is the burying beetle species *Nicrophorus vespilloides*. Like other members from this genus, *Nicrophorus*

uses carcasses of small vertebrates both as a food resource and as a nursery for their progeny (Eggert *et al.* 1998; Scott 1998). In the wild, when a male and female pair locate a carcass, they strip away any outer layers of fur or skin and bury it underground in order to avoid competition from conspecifics and competitors (Eggert *et al.* 1998). The female then lays her eggs around the carcass, which when hatched, begin to self-feed or are provisioned by the parents until independence from care about five days later. After three weeks, the larvae pupate into adults and are sexually mature after eleven days. Both parents may be involved in the parental care of the larvae but, typically, the male abandons the brood prior to larval dispersal from the carcass (Bartlett 1988; Scott & Gladstein 1993). Importantly, studies have shown that the experimental removal of one parent in the laboratory, in order to study the individual effects of paternity or maternity, does not detrimentally impact offspring performance (Smiseth *et al.* 2005). Additionally, burying beetle larvae can be cross-fostered without consequence if the timing of maternal reproduction is synchronised between mothers (Müller & Eggert 1990). For this reason the burying beetle makes an ideal study system to investigate the pre- and postnatal components of maternal effect ageing (Lock *et al.* 2004, 2007; Head *et al.* 2012).

1.5.1 Nicrophorus vespilloides ageing experiments

Despite a number of key life history traits that make the *Nicrophorus* an amenable genus to study patterns of senescence, the field of *Nicrophorus* ageing research is relatively sparse. On ten previous occasions has this genus been used to explore some manifestation of ageing (Lock *et al.* 2007; Creighton *et al.* 2009; Trumbo 2009, 2012;

Ward *et al.* 2009; Cotter *et al.* 2010; Benowitz *et al.* 2013; Billman *et al.* 2014; Lee *et al.* 2014; Takata *et al.* 2016). Of these, only five use the *Nicrophorus vespilloides* (Lock *et al.* 2007; Ward *et al.* 2009; Cotter *et al.* 2010; Benowitz *et al.* 2013; Lee *et al.* 2014), and only three investigate the detrimental effects of increasing maternal age (Lock *et al.* 2007; Ward *et al.* 2009; Cotter *et al.* 2010). In fact, very few *Nicrophorus* ageing experiments have been conducted that test the individual consequence of age whilst correcting for differences in parity. Lock *et al.* (2007) experimentally separated the effects of pre- and postnatal maternal age and investigated whether any reduction in offspring quality (as a result of declining prenatal maternal effects) could be mitigated by adaptive changes in postnatal care. They found convincing evidence for coadaptation between both pre- and postnatal maternal care, with females able to adjust their behaviour in response to maternal-age-related changes in offspring quality. Ward *et al.* (2009) and Cotter *et al.* (2010) investigated the combined effects of maternal age and repeated parity (i.e. without the use of cross-fostering to separate pre- and postnatal components) on the number and size of dispersing larvae. In particular, research by Cotter *et al.* (2010) provided convincing evidence of declining total weight of offspring with increasing first age at reproduction (from 24 to 60 days). However, Ward *et al.* (2009) confounded the effect of multiple breeding attempts and increasing age by performing successive reproductive bouts every 12 days. Ostensibly we could therefore expect that any observed declines could represent incurred reproductive and physiological costs of multiple matings rather than a true senescent signature. Additionally, potential bias associated with selective disappearance, could potentially be contributing to unclear measures of senescence (van de Pol & Verhulst 2006; van de Pol & Wright 2009; Nussey *et al.* 2011). As mentioned previous, the addition of longevity as a statistical covariate or

the use of within-between subject centring is commonly used to account for such bias (van de Pol & Wright 2009). However, no studies have investigated the transgenerational effects of age in the genus *Nicrophorus* whilst also accounting for the problematic issue of demographic heterogeneity affecting maternal age effect estimates.

1.6 Thesis Aims

This thesis addressed the following outstanding questions relating to senescence and maternal effect ageing:

1.6.1 How are maternal age effects distributed across multiple taxa?

Using both experimental (Chapter 2) and meta-analytical (Chapter 3) techniques I explored the distribution and direction of maternal age effects across multiple animal taxa. In particular, I explored the decoupled effects of increasing pre- and postnatal maternal age through experimentally cross-fostered broods of the burying beetle *Nicrophorus vespilloides,* whilst accounting for the potential bias of postnatal selective disappearance (Section 1.3.1). Furthermore, as stated in Section 1.1.1, whilst these maternal age effects are becoming increasingly more discussed, the distribution of these effects is unknown (Bloch Qazi *et al.* 2017). To this end, I employed meta-analytical techniques to extensively investigate the distribution of these maternal age effects on a broader scale, and compare across multiple animal taxa and contrasting environments. In particular, we hoped to identify whether maternal age was an important determinant of offspring performance, specifically neonatal survival, throughout the tree of life.

Taken together, the experimental technique employed in Chapter 2 represents the most appropriate empirical design to date for measuring the individual contributions of maternal age on neonatal survival whilst also accounting for the bias of selective disappearance. This chapter serves as a model design for comparison with other research into maternal age effects, extensively reviewed in Chapter 3.

1.6.2 How does a knowledge of natural selection and evolution help us to understand the observed diversity in maternal age effects and demographic senescence?

In order to better understand if evolution by natural selection has helped shape the enormous variation in observable trait diversity that is exhibited throughout the tree-of-life, it is necessary to test the assumptions and predictions of classical evolutionary theory. This was investigated using two differing techniques: 1) Evolutionary theory focusing on maternal effect senescence by Moorad and Nussey (2016) (Chapter 3) was assessed in its ability to describe patterns of maternal effect ageing across a multitude of animal taxa. In particular, we wanted to identify whether the performance of this evolutionary theory improved when describing maternal effect ageing in natural, wild populations in comparison to those kept under artificial conditions in the laboratory. This allowed us to ask to what extent was natural selection contributing to evolution of maternal effect senescence; 2) Classical evolutionary theory by Hamilton (1966) (Chapter 4) was assessed in its ability to predict vital rate ageing trajectories across multiple wild populations. In particular, by exploring the relative distribution of violations from Hamilton-like ageing and identifying whether these departures were more likely to occur in either age-specific fertility or mortality.

Describing the distribution of these violations would then allow us to: 1) identify whether natural selection can accurately describe patterns of vital rate ageing, in doing so also evaluate the evolutionary theory put forward by Hamilton (1966); 2) highlight potential taxonomic hotspots for violations; and 3) highlight potential mechanistic causes or biological processes that are readily contributing to departures from basic theory.

Additionally, as eluded to in Section 1.3, a number of life history processes or constraints exist that can lead to departures from patterns of ageing predicted by evolutionary theory. These processes are explored in depth within Chapters 2-4, with particular focus placed upon accounting and testing for the effects of: 1) positive genetic correlations across ages, or selective disappearance (Section 1.3.1); 2) increases in offspring performance due to reproductive effort and the terminal investment hypothesis (Section 1.3.2) and 3) environmental variation between laboratory and natural populations (Section 1.1.3).

In summary, Chapters 2-4 aim to assess the overall value of the evolutionary theory of senescence and the performance of natural selection in predicting patterns of ageing, as it pertains to vital rates and maternal effects, in real populations of animal species existing both in natural and laboratory environments.

Chapter 2: Disentangling pre- and postnatal maternal age effects on offspring performance in an insect with elaborate maternal care

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2.1 Introduction

Senescence is often described as an age-related physiological deterioration associated with increasing mortality risk and decreasing reproductive rates (Finch *et al.* 1990; Jones *et al.* 2014). The deleterious effect of changes on individuals' own fitness-related traits has long been explained by the evolutionary theory of ageing (Williams 1957; Hamilton 1966). However, maternal effect senescence, the detrimental effects of increasing maternal age that are manifested in the traits of their offspring, is a fundamentally different form of ageing that has attracted recent interest (Heidinger *et al.* 2016; Moorad & Nussey 2016; Warner *et al.* 2016; Lippens *et al.* 2017). The most noted offspring outcomes are reduced offspring juvenile survival (Hercus & Hoffmann 2000; Sharp & Clutton-Brock 2010) and adult longevity (Lansing 1947; Priest *et al.* 2002; Fox *et al.* 2003). Evolutionary theory predicts deleterious effects of maternal ageing on early offspring survival accompanied by an age-related increase in genetic variance for maternal effects (Moorad & Nussey 2016), but no such evolutionary model has been developed to explain the negative maternal age effect on offspring adult longevity. However, this phenomenon has been observed frequently enough to be given a term, the 'Lansing effect', after an early observer Albert Lansing (Comfort 1953).

Some life history theory predicts that reproductive effort, or the proportion of available resources allocated to reproductive processes, should increase with age as the potential to realise future costs to reproduction lessens (Williams 1966b; Charlesworth & Leon 1976; Clutton-Brock 1984). This is expected to mitigate the observed expression of reproductive senescence. Reproductive effort has been reported to increase with age in several taxa, including species of birds (Pugesek 1981; Velando *et al.* 2006), mammals

(Ericsson *et al.* 2001; Paterson *et al.* 2016), fish (Poizat *et al.* 1999) and invertebrates (Kight *et al.* 2000; Creighton *et al.* 2009; Hargrove *et al.* 2018). In species that provide elaborate parental care, it has been argued that the increased reproductive effort is manifested as larger and more successful offspring at the cost of reduced parental condition (Bijleveld & Mullers 2009). However, many studies fail to find age-related increases (Dugdale *et al.* 2011; Martin & Festa-Bianchet 2011; Conover 2013; Kuczynski *et al.* 2015).

Age-related changes in maternal effects can be caused by altered prenatal (e.g., egg-mediated) and postnatal (e.g., care-mediated) contributions to offspring condition (Beamonte-Barrientos *et al.* 2010; Lemaître & Gaillard 2017). However, distinguishing between the two can be extremely difficult in many systems because the providers of prenatal and postnatal care are often the same individuals (Lock *et al.* 2007; Beamonte-Barrientos *et al.* 2010). Nevertheless, it is important to decouple these effects in order to understand better the proximate mechanisms of maternal effect changes with age that are mediated through changes in egg quality or rearing capabilities (Beamonte-Barrientos *et al.* 2010). In species with complex parental care, it is important to consider both aspects of maternal effects, as pre- and postnatal age can each have large influence on offspring fitness (Lock *et al.* 2007). Cross-fostering, where offspring born to one parent are raised by another, is a useful method for decoupling these two effects (Lynch & Walsh 1998). Applied to studies of senescence, cross-fostering offers a way to reveal potential divergent age-related effects of pre- and postnatal maternal effects. This approach has been used to study maternal age effects in the blue-footed booby, *Sula nebouxii*, where both the age of egg-producers and carers were found to independently

decrease growth rates of second chicks (laying-mother age (years): β = -1.00 mm/day, rearing-mother age (years): β = -1.80 mm/day; Beamonte-Barrientos *et al.* 2010).

Whilst evolutionary models predict age-related changes in individual traits, demographic models suggest that among-individual variation (individual heterogeneity) can cause age-specific means for fitness related traits to increase due to selective disappearance of frail individuals (Vaupel *et al.* 1979; Vaupel & Yashin 1985; van de Pol & Verhulst 2006). As a result, age-related changes in trait values may reflect changes in the identities of the individuals that make up the population rather than longitudinal changes in individuals caused by ageing. This phenomenon has been shown to influence the perceived effects of age on fitness traits in various mammal and bird species (Cam *et al.* 2002; Nussey *et al.* 2006, 2011). Fortunately, the effects of selective disappearance can be corrected for statistically in longitudinal studies of ageing (van de Pol & Verhulst 2006; van de Pol & Wright 2009; Nussey *et al.* 2011). If left uncorrected, inferences about of the effects of ageing on individuals may be unreliable (Hayward *et al.* 2013). Controlling for the effects of heterogeneity has only seldom been applied to maternal effect ageing research (e.g. Hayward *et al.* 2013; Berger *et al.* 2015; Fay *et al.* 2016a). There is a general need for an integration of methodologies that combine cross-fostering with the ability to control for selective disappearance.

We performed an ageing experiment with a species of burying beetle, *Nicrophorus vespilloides*, with conspicuous postnatal maternal care. This species provides an ideal study system for discriminating between pre-and postnatal maternal effects as larvae can be cross-fostered (Lock *et al.* 2004, 2007; Head *et al.* 2012). Furthermore, whilst both parents can simultaneously provide care, the removal of one parent does not detrimentally effect average larval body weight or survival in the laboratory (Smiseth *et*

al. 2005). Using a cross-fostered experimental design, we aimed to decouple the prenatal (egg-producer) and postnatal (carer) effects of different maternal ages on life history traits of their offspring and fitness-related outcomes of care-giving females. Existing models of maternal effect senescence (Moorad & Nussey 2016) predict that as maternal age increases into late age, we should see corresponding declines in offspring survival. This theory makes no explicit predictions regarding other offspring traits, but it is reasonable to predict that similar evolutionary arguments should predict that offspring adult longevity and larval dispersal weight will decrease with increasing maternal age. Previous *N. vespilloides* research is consistent with this expectation by showing that increased maternal age reduced the number of hatched larvae (Creighton *et al.* 2009), dispersed larvae and total weight of brood at dispersal (Ward *et al.* 2009; Cotter *et al.* 2010). We may also expect that old egg-producers will negatively impact the life history of the carer if they produce low quality offspring. In fact, cross-fostered *N. vespilloides* females have been shown to provide more care when given lower quality larvae to care for (Mattey *et al.* 2018), and it's possible that providing this additional care comes at a cost. Lastly, we corrected for selective disappearance statistically by adding carer longevity as a factor to our analyses of postnatal effects; this is a novel approach to ageing research into postnatal maternal effects in a controlled laboratory population.

2.2 Materials and Methods

2.2.1 Study System

N. vespilloides breed and feed on small, dead vertebrates. Breeding pairs prepare the carcass by removing all hair, feathers, or scales and roll the carrion into a ball before

burying it in the soil (Scott 1998). The female lays eggs in the surrounding soil. Two to three days later, the eggs hatch and the larvae move to the carcass where they can self-feed and be provisioned and cared for by their parents until independence, which occurs after four or five more days. Parental care in this species is characterized by the regurgitation of carrion from parents to larvae, defence of the larvae and carcass from conspecifics and other competing species, and the secretion of an anal exudate that inhibits fungal growth on the carcass. Larvae disperse into the surrounding soil after they become independent from parental care and pupate into adults (eclose) 21 days later.

The beetles used in this study were taken from an outbred laboratory population maintained at the University of Edinburgh originally derived from a colony in the Netherlands kindly provided by Daniel Rozen in 2013. Genetic diversity has since been maintained and enhanced by annual additions of wild beetles trapped from natural populations around Edinburgh. Beetles were individually housed in clear plastic boxes, kept at 21°C at a 16 hours light: 8 hours dark cycle and fed small pieces of organic beef twice a week.

2.2.2 Experimental Age Classes

Female beetles were sampled from the population at four different post-eclosion age ranges: "Young", "Mid-Life", "Old" and "Very Old" (comprising 11-18, 32-39, 53-60, and 77-87 days respectively). Females are seldom reproductively active before ten days post-eclosion (Cotter *et al.* 2010), and female virgins that are older than "Very Old" are exceedingly rare. Female ages were also selected to represent differing rates of

cumulative survival in virgin females (94%, 80%, 26% and 1%) (Moorad, unpublished data) and thus presumably represent highly varied magnitudes of selection for age-specific maternal care that covers nearly the full potential lifespan of the beetle (Moorad & Nussey 2016). We used virgin beetles for two reasons. First, differences among individuals in past reproductive allocation could contribute unnecessarily to trait variance even if the previous number of reproductive events was considered as a correlate in the statistical models. Second, with multiple matings, female age will be necessarily confounded with reproductive history, and the strong correlation between age and mating experience can cause additional statistical problems. One such problem could be that reproductive experience and increased age both have negative effects, and conflating the two will overestimate the true effects of age.

2.2.3 Experimental Procedures

We used a cross-fostered design to assess offspring performance in relation to varying carer and egg-producer age. Virgin females from the four age classes were mated with virgin males aged approximately two weeks post-eclosion. The male ages were standardized in order to reduce variation caused by effects of paternal age. We supplied each pair with a mouse carcass weighing 20.71 – 25.99 g (Livefood Direct Ltd, Sheffield, UK). Females were weighed before breeding, after egg laying and after providing care. Males were removed 72 hours after introduction to the carcasses and mating, and females with carcasses were placed into new breeding containers (absent of any eggs or larvae) in preparation for them to receive a mixed brood of larvae from other females. The old mating boxes (those which had previously contained females, carcasses and

eggs) were checked for newly hatched larvae every two hours for five minutes until no new larvae were found (~72 hours). Females that recently produced hatched larvae are capable of caring for other larvae, provided that these have hatched at roughly the time point as their own. If larvae appear on the carcass too early or too late, females will perceive them as not theirs and kill them (Müller & Eggert 1990). Infanticide from cross-fostering did not appear to have occurred in this experiment as no mothers eliminated whole broods of larvae. Previous work involving several *Nicrophorus* species used similar cross-fostering techniques (Rauter & Moore 2002a; Lock *et al.* 2004, 2007; Head *et al.* 2012; Steiger 2013).

We pooled larvae from same-age mothers (Rauter & Moore 2002b; Crook *et al.* 2008; Rozen 2008; Arce *et al.* 2012). *N. vespilloides* females produce highly variable brood sizes (Smiseth & Moore 2002), and considerable asynchrony in larval hatching is frequently observed (Smiseth *et al.* 2008; Ford & Smiseth 2016). Pooling larvae produced by different mothers into mixed broods was a tractable approach to generating suitable numbers of experimental broods with constant family size. From these, we randomly sampled larvae to construct mixed broods of 15 larvae each to control for initial effects of density. A brood size of 15 larvae struck a reasonable balance between obtaining sufficient numbers of mixed broods whilst falling within the range of normal brood sizes produced by *N. vespilloides* (range 2-47) (Smiseth & Moore 2002; Smiseth & Parker 2008). Each mixed brood was placed under the care of unrelated mothers of various age classes and allowed to develop in the presence of their foster mother (the carer). At dispersal, larvae were counted and individually weighed using an Ohaus Pioneer PA114 analytical balance (repeatability = 0.1mg). Mated females and eclosed offspring were then individually housed, regularly fed (with raw organic beef twice a week) and checked

for death (three times a week) until all beetles had died. In total, we set up 147 matings, with 55 females caring for a brood (Table 2.1). The other 92 females provided no care and either donated larvae after mating (n = 55), had eggs that did not hatch (n = 15) or were omitted as the matings and females could not be used (n = 22).

Table 2.1 Numbers of care-giving and egg-producing beetles in each experimental age class.

Age class	Young egg-producer	Mid-life egg-producer	Old egg-producer	Very old egg-producer
Young carer	5 (75)	6 (90)	6 (90)	5 (75)
Mid-life carer	8 (120)	2 (30)	3 (45)	-
Old carer	5 (75)	6 (90)	4 (60)	-
Very-old carer	5 (75)	-	-	-

Note - Numbers of larvae are given in parentheses.

2.2.4 Statistical Analyses

We used ASReml v.4.1 (Gilmour 1997) to construct univariate generalised linear mixed effect models using data observed at the level of the offspring (Table 2.2) to independently measure both carer age and egg-producer age effects upon larval weight at dispersal and offspring adult longevity (both with Gaussian error structures). Next, we fit a multivariate mixed effect model using data that were collected at the level of the carer to evaluate the effects of the care-givers' and egg-producers' age upon traits related to fitness. Dependent variables were larval weight at hatching (pooled over all broods of 15 larvae), residual lifespan of carer (days survived post-mating), carer weight

change (difference between post-care and pre-care body weight) and number of larvae surviving to dispersal. Note that the two univariate models fit using data collected at the level of the offspring feature offspring outcome traits as dependent variables, whilst the multivariate model was fit using data collected at the level of the carer features dependent variables that describe both offspring and carer traits. All models were fit twice: once with first-order effects of carer and egg-producer age and once with all three possible second-order interactions involving these ages.

Parameters with the potential to confound the relationships between maternal ages and offspring outcomes were included in the mixed models as fixed and random effects. These fixed effects were carcass size, as resource availability is known to effect larval fitness (Trumbo 1990), and carer age at death, in order to account for selective disappearance that might otherwise mask signatures of senescence (van de Pol & Verhulst 2006). When used as a predictor, age at death was defined according to the interval at which the event occurred (added as a four-level factor to identify age intervals between reproductive opportunities: between "Young" and "Mid-life", between "Mid-life" and "Old", between "Old" and "Very Old", and beyond "Very Old"). In longitudinal studies, the effects of selective disappearance are usually modelled by fitting linear or quadratic functions of longevity (Bouwhuis *et al.* 2009; Millon *et al.* 2011; Nussey *et al.* 2011; Hayward *et al.* 2013). This practice is appropriate when phenotypic observations and deaths are distributed continuously over ages, but it is not the best approach for analyses of controlled experiments in which phenotypes are collected in relatively few discrete age ranges at regular intervals. There are two reasons for this. First, the categorical approach used here accommodates more complex age functions (three parameters originating from four age intervals vs two parameters from a quadratic function). Second,

variation in age at death that occurs within intervals (e.g., different ages at death that all occur between “Young” and “Mid-Life”) or outside of the intervals (different ages of death after “Very Old”) should not contribute to model fitting. This constraint is appropriate because all individuals that die within intervals are all equally dead at the onset of the next age class, and their precise timing of removal within intervals should be non-informative. In this case, implementing age as a continuous effect (as is often done in studies of natural populations) would inappropriately allow within-interval variation to influence parameter estimates. Our experiment allowed us to correct for the effects of selective disappearance of care-givers but not egg-producers, as the mixed-brood design made the identification of egg-producers impossible.

The effects of block (as the experiment was split into nine experimental blocks) and carer IDs were added as nested random effects (carer ID nested within block). The latter was included to account for possible effects of pseudo-replication as individual mothers care for multiple offspring. The full multivariate model that fit linear age effects failed to converge, indicating a non-positive-definite variance covariance structure for block effects. We ran univariate analyses for each female trait with and without the random effect of block to learn if we could justify dropping block effects from the full model. The *p* values from a likelihood ratio test between the two models were as follows: larval weight at hatching p=0.240; carer residual lifespan p=0.357; carer weight change p=1.000 and number of larvae surviving to dispersal p=0.031 (see Table S2.1 for likelihood ratio test results). These results justified rerunning the unconstrained full model whilst including block effects only for the number of larvae surviving to dispersal.

For each of the six traits of interest, comparisons were made between three models that fit maternal age effects in different ways: 1) no effect of carer and egg-

producer age (the null model); 2) first-order effects of carer and egg-producer age (the linear model); and 3) all first and second-order effects of both ages (the quadratic model). As restricted maximum likelihood methods are not appropriate for model comparisons, maximum likelihood was used instead. For larval weight at hatching, residual lifespan, and carer weight change, there was no need to fit random effects in the model because: 1) observations were made at the level of the carer (no carer ID effects), and 2) block effects were shown to be statistically insignificant (no block effect). Consequently, general linear models were applied using R v.3.3.3 (R Core Team 2016). The other traits required models that included random effects: all models required block effects, and offspring longevity and dispersal weight required carer ID effects. For these traits, we applied a mixed model approach using "lme4" v.1.1-15 (Bates *et al.* 2015). Models were compared using Akaike Information Criterion (AIC) values.

Table 2.2 Summary of mixed models

Level	Response Variable	Full Model
Offspring	Larval weight at dispersal	Fixed Effects: Carer age + egg-producer age + carcass weight + age of carer at death Random Effects: Block/carer ID
Offspring	Offspring adult longevity	Fixed Effects: Carer age + egg-producer age + carcass weight + age of carer at death Random Effects: Block/carer ID

Carer	Larval weight at hatching Residual lifespan of carer Weight change of carer Number of larvae surviving to dispersal	Fixed Effects: Carer age + egg-producer age + carcass weight + age of carer at death Random Effects: Block (for trait 4)

Note - Quadratic forms of the models add all possible second-order interactions involving carer age and egg-producer age (two squared terms and one cross-product).

Table 2.3 Summary of AIC comparison for model selection

Traits	Null	Linear	Quadratic
Larval weight at dispersal	-2644.117	-2642.683	**-2647.966**
Offspring adult longevity	**4911.746**	4914.960	4915.163
Larval weight at hatching	**-372.415**	-370.567	-365.987
Residual lifespan of carer	513.974	**485.546**	486.427
Weight change of carer	**-211.259**	-210.798	-205.476
Number of larvae surviving to dispersal	273.494	274.992	**269.105**

Note – The model with the lowest AIC is presented in bold-face.

2.3 Results

Two carers were lost post-care, but their offsprings' data have been included in the offspring-level analyses because post-care maternal longevity had no detectable effect on offspring outcomes (see below). These females were excluded from the multivariate analysis. Model selection indicated that: 1) null models were best for describing offspring adult longevity, larval weight at hatching and female weight; 2) a linear model best described residual lifespan of the carer; and 3) quadratic models best

described larval weight at dispersal and number of larvae surviving to dispersal (Table 2.3, see Table S2.2 for full model selection comparison.).

2.3.1 Larval weight at dispersal

In the linear model, egg-producer age was shown to have a negative effect on larval weight at dispersal whilst carer age was shown to have a positive effect. However, neither effect was statistically significant (Table 2.4, Fig. S2.1(a)). In the quadratic model, there were detectable positive linear and negative quadratic effects of egg-producer age. Carer age had no detectable effects on larval weight at dispersal (Table 2.4, Fig. S2.1(b)). There was a statistically significant negative effect of age of carer at death (2 to 5 weeks) on larval weight at dispersal in the quadratic model. No other measured covariates affected larval dispersing weight.

Table 2.4 Effect of age on larval weight at dispersal

Model	Covariate	Effect Size Estimates *mg/day*	Standard Errors *mg/day*	*z* score	*p* value
Linear	Carer age	0.152	0.235	0.647	0.518
	Egg-producer age	-0.239	0.206	-1.159	0.246
	Carcass weight	3.466	3.036	1.142	0.254
	Age of carer at death (2 to 5 weeks)	-49.780	28.720	-1.733	0.083
	Age of carer at death (5 to 8 weeks)	9.806	20.180	0.486	0.627

	Age of carer at death (8 to 11 weeks)	4.275	12.600	0.339	0.734
	Age of carer at death (11+)	0.000	0.000	0.000	-
	Carer age	0.459	1.180	0.389	0.697
	Egg-producer age	**2.690**	**1.201**	**2.240**	**0.025**
	Carer age*egg-producer age	-0.007	0.014	-0.514	0.607
	Carer age^2	-0.001	0.011	-0.117	0.907
	Egg-producer age^2	**-0.034**	**0.012**	**-2.934**	**0.0033**
	Carcass weight	3.296	2.940	1.121	0.262
Quadratic	**Age of carer at death (2 to 5 weeks)**	**-57.020**	**28.890**	**-1.974**	**0.048**
	Age of carer at death (5 to 8 weeks)	24.220	20.640	1.173	0.241
	Age of carer at death (8 to 11 weeks)	5.559	11.380	0.488	0.625
	Age of carer at death (11+)	0.000	0.000	0.000	-

Note- *z*-scores were derived by dividing effect sizes by standard errors, and *p* values were calculated from these. Effects that were significant to a threshold of $\alpha = 0.05$ are in bold-face.

2.3.2 Offspring adult longevity

In the linear model, egg-producer age was shown to have a negative effect on offspring adult lifespan whilst carer age was shown to have a positive effect, but neither were statistically significant (Table 2.5, Fig. S2.22(a)). No linear or quadratic effects of egg-producer and carer age on offspring adult lifespan (Table 2.5, Fig. S2.2(b)) were detected

in the quadratic model. No other measured covariates affected offspring adult longevity in either model.

Table 2.5 Effect of age on offspring adult longevity

Model	Covariate	Effect Size Estimates *Lifespan/day*	Standard Errors *Lifespan/day*	*z* score	*p* value
Linear	Carer age	0.001	0.124	0.006	0.995
	Egg-producer age	-0.075	0.112	-0.675	0.500
	Carcass weight	0.350	1.566	0.223	0.823
	Age of carer at death (2 to 5 weeks)	-6.120	14.690	-0.417	0.677
	Age of carer at death (5 to 8 weeks)	-4.700	11.140	-0.422	0.673
	Age of carer at death (8 to 11 weeks)	-3.956	6.641	-0.596	0.551
	Age of carer at death (11+)	0.000	0.000	0.000	-
Quadratic	Carer age	0.777	0.635	1.225	0.221
	Egg-producer age	-0.247	0.654	-0.378	0.705
	Carer age*egg-producer age	-0.010	0.007	-1.326	0.185
	Carer age^2	-0.006	0.006	-1.039	0.299
	Egg-producer age^2	0.006	0.007	0.883	0.377
	Carcass weight	0.348	1.547	0.225	0.822

	Age of carer at death (2 to 5 weeks)	-6.827	14.940	-0.457	0.648
	Age of carer at death (5 to 8 weeks)	-6.993	11.680	-0.599	0.549
	Age of carer at death (8 to 11 weeks)	-3.370	6.425	-0.525	0.600
	Age of carer at death (11+)	0.000	0.000	0.000	-

Note - *z*-scores were derived by dividing effect sizes by standard errors, and *p* values were calculated from these.

2.3.3 Traits assessed at the level of the carers

In the linear model, neither carer age nor egg-producer age affected any of the measured carer-level traits (Table 2.6, Fig. S2.3(a)-S2.6(a)), with the sole exception of a negative relationship between carer age and post-care residual lifespan. Incidentally, we should not expect any meaningful effects of carer age on larval weight at hatching because this experimental design ensures that carers do not influence offspring development until after hatching. In the quadratic model, there was neither linear nor quadratic effects of carer age on any of the measured carer-level traits (Fig. S2.3(b)-S2.6(b)). However, egg-producer age had a positive linear and a negative quadratic effect of on the number of larvae surviving to dispersal (Table 2.6 and Table S2.3(b)). Furthermore, carer age and egg-producer age interacted to cause a statistically significant negative effect on this trait. Age of carer at death (2 to 5 and 8 to 11) affected the residual lifespan of the carer

in both the linear and quadratic models. No other measured covariates affected the measured traits at the level of the carer (see Table S2.3(a) and S2.3(b)).

Table 2.6 Effect of age on various offspring and carer outcomes

Model	Female Age	Trait	Effect Size Estimate Unit/day	Standard Error Unit/day	*z* score	*p* value
Linear	Carer	Larval weight at hatching	-0.077	0.056	-1.363	0.173
		Residual lifespan of carer	**-1.122**	**0.182**	**-6.182**	**<0.001**
		Weight change of carer	0.394	0.255	1.549	0.121
		Number of larvae surviving to dispersal	-0.032	0.025	-1.259	0.208
	Egg-producer	Larval weight at hatching	-0.035	0.051	-0.688	0.491
		Residual lifespan of carer	-0.107	0.163	-0.656	0.512
		Weight change of carer	-0.097	0.229	-0.425	0.671
		Number of larvae surviving to dispersal	-0.028	0.023	-1.209	0.228
	Carer	Larval weight at hatching	0.0385	0.3201	0.120	0.904
		Residual lifespan of carer	-1.163	0.995	-1.169	0.242
		Weight change of carer	-0.0309	1.4550	-0.021	0.983

		Number of larvae surviving to dispersal	0.220	0.120	1.832	0.067
Quadratic	Egg-producer	Larval weight at hatching	0.2667	0.3224	0.827	0.408
		Residual lifespan of carer	1.356	1.002	1.353	0.176
		Weight change of carer	0.1095	1.4660	0.075	0.940
		Number of larvae surviving to dispersal	**0.319**	**0.121**	**2.643**	**0.008**
	Carer^2	Larval weight at hatching	-0.0003	0.0029	-0.108	0.914
		Residual lifespan of carer	0.0049	0.0089	0.550	0.582
		Weight change of carer	0.0060	0.0131	0.462	0.644
		Number of larvae surviving to dispersal	-0.0017	0.0011	-1.509	0.131
	Egg-producer^2	Larval weight at hatching	-0.0025	0.0031	-0.823	0.411
		Residual lifespan of carer	-0.0129	0.0095	-1.358	0.174
		Weight change of carer	-0.0012	0.0139	-0.089	0.929

	Number of larvae surviving to dispersal	**-0.0028**	**0.0012**	**-2.300**	**0.021**
	Larval weight at hatching	-0.0033	0.0037	-0.887	0.375
	Residual lifespan of carer	-0.014	0.011	-1.214	0.225
Carer*Egg-producer	Weight change of carer	-0.0033	0.0168	-0.199	0.842
	Number of larvae surviving to dispersal	**-0.004**	**0.001**	**-3.065**	**0.0022**

Note - Units are milligrams for weight and weight change measurements, days for residual lifespan, and counts for larvae numbers. *z*-scores were derived by dividing effect sizes by standard errors, and *p* values were calculated from these. Effects that were significant to a threshold of $\alpha = 0.05$ are in bold-face.

2.4 Discussion

For three of the six traits investigated here (offspring adult longevity, larval weight at hatching, and weight change of carer), model comparisons and estimated effect sizes clearly indicate the absence of significant maternal age effects. Our results show a clearly negative effect of carer age on the residual lifespan of carers, but this simply reflects actuarial senescence, or an increase in mortality risk with increasing age (Finch *et al.* 1990). If older egg producers generate lower quality offspring and these place a greater burden on older care-givers, then we might expect a negative interaction between the ages of egg-producers and carers. Whilst we did estimate a negative interaction effect, it was not statistically significant. For two traits, larval weight at dispersal and number of survived larvae, model selection indicated that the quadratic models were best, and the linear models were worst. In both cases, quadratic estimates suggest convex relationships between larval outcomes and egg-producer age (there was also a negative interaction effect on larval survival between the ages of egg-producers and carers). This quadratic pattern to ageing has been observed in both mammal (Weladji *et al.* 2002; Nussey *et al.* 2006; Dugdale *et al.* 2011; Linares 2013; English *et al.* 2014) and bird species (Bouwhuis *et al.* 2009, 2010; Torres *et al.* 2011; Drummond & Rodríguez 2015). However, there are reasons to view our results with scepticism. First, we estimate a large number of quadratic effects (three effects for each of the six traits), and we expect some estimates may be statistically significant owing only to chance. It might be appropriate in this case to correct for multiple comparisons. A Bonferroni correction (Bonferroni 1936) reduces the threshold for rejecting the null model of no quadratic effect to $\alpha = 0.0028$. The effects of egg-producer age^2 fail to reach this threshold, but the

interaction of carer age and egg-producer age on number of larvae surviving to dispersal satisfies this condition. We note that the statistically significant effect of carer age on residual carer lifespan (actuarial senescence) remains after a Bonferroni correction (12 estimated linear age effects, α = 0.0042). Second, we must be circumspect when interpreting quadratic effects of egg-producer age because our experimental design did not allow us to correct for selective disappearance of egg-producers. This phenomenon is discussed more generally below, but applied to this situation, we would expect that the preferential removal of poor mothers early in life could lead to the observed concave functions of larval weight and survival against age. Given a lack of meaningful linear maternal effects and only ambiguous quadratic effects, a conservative explanation of our results is that we failed to detect pre- and postnatal maternal effect senescence for several traits related to fitness.

As other work on *Nicrophorus* species has detected changes in offspring outcomes with maternal age, it is important to carefully evaluate our negative findings against these with a particular emphasis placed upon assessing the relative statistical power of our analysis. To interpret statistically non-significant results, evaluating the effect size and their relative confidence intervals is preferred to post-hoc power analyses (Colegrave & Ruxton 2003). We do this by asking if effect sizes reported in previous *N. vespilloides* studies fall within the 95% confidence intervals estimated here using linear models. However, some comparisons are impossible because our study was the first to evaluate the effects of maternal age on many traits. Furthermore, previous studies very rarely report effect size estimates (preferring instead to emphasize significance). Consequently, in some cases, we had to estimate previous effect sizes using age-class data presented in graphical form in the source papers (see Supplementary Table S2.4 for

details). Ward *et al.* (2009) reported that larval dispersal weight changed by -1.97mg/day of maternal age for multiply-mated females. Cotter *et al.* (2010) surveyed brood weights at dispersal from virgin females bred at different ages, but they did not report larval number in their analysis. This study observed a total brood weight change of -30mg/day of maternal age. A generous larval density estimate of 1.88g/day (see Smiseth & Moore 2002) suggests a per-larval effect size of -1.60mg/day of maternal age. Both of these estimates lie far beyond the 95% confidence intervals estimated here for egg-producer and carer age effects ([-0.64mg/day, +0.16mg/day] and [-0.31mg/day, +0.61mg/day], respectively; Table S2.4). The Ward *et al.* study reported that larval survival declined 0.0087 1/day of maternal age; the estimated 95% confidence intervals for this effect here was [-0.0048 1/day, +0.0011 1/day] and [-0.0054 1/day, +0.0012 1/day] for the two maternal age effects (see estimates in Table 2.6 divided by 15 for the initial brood size). In general, Type 2 errors can always be a concern with reports of negative results, but these comparisons make clear that any true effect in our population is much smaller than other published estimates from previous comparable studies.

Why are the effect sizes reported here so much smaller than in other *Nicrophorus* studies? This may have been the result of important improvements in our experimental design that enabled us to estimate the true effects of maternal age with more rigour. Unlike other ageing experiments in *Nicrophorus* sp., this experiment used virgin females. Whilst these may be difficult to obtain, especially in the older age classes (owing to ever-decreasing rates of cumulative survival), using virgins in all age classes removes the risk of conflating the effects of breeding experience with the effect of maternal age. Secondly, this experiment successfully accounted for potential bias attributed to selective disappearance. However, no contributions of selective disappearance of carers

to perceived ageing patterns for any offspring trait was found. This suggests that any non-random subset of these females that survived to old age did not bias our results by producing higher quality offspring with higher larval dispersal weight and longevity. Heterogeneity appears to be ubiquitous in wild vertebrates systems (Nussey *et al.* 2011), where it appeared to act to obscure evidence of maternal effect senescence in at least one study (Hayward *et al.* 2013). It is unknown whether heterogeneity may have influenced the results from other laboratory *Nicrophorus* studies of ageing. We note that we were unable to test for effects of selective disappearance of egg-producers because this study used mixed broods, which unfortunately prohibited adding egg-producer longevity to the models. Future research should focus on fully accounting for the effects of the selective disappearance of these mothers on offspring life-history traits. Using intact broods of larvae, where the egg-producers' identities are known, would allow us to completely account for the effects of heterogeneity upon both maternal influences.

Neither the evolutionary predictions made by Moorad and Nussey's (2016) ageing models nor those from reproductive effort models (Williams 1966a; Hirshfield & Tinkle 1975; Charlesworth & Leon 1976; Clutton-Brock 1984) applied to maternal effects on larval survival are supported by our results. Other studies show mixed evidence for maternal age effects on juvenile survival. Some show declines, such as in *Panthera pardus* (Balme *et al.* 2013), *Papio anubis* (Packer, Tatar & Collins 1998), *Panthera leo* (Packer *et al.* 1998), *Ovis aries* (Hayward *et al.* 2013, 2015) and *Parus major* (Perrins & Moss 2008). Whilst others show no effect of age, such as in *Glossina palpalis palpalis* (McIntyre & Gooding 1998), *Podisus maculiventris* (Mohaghegh *et al.* 1998), *Nauphoeta cinerea* (Moore & Harris 2003) or an increase, *Vanellus vanellus* (Blomqvist *et al.* 1997). Why this variation

exists and how it is distributed across species is unclear, and these questions deserve future study. Unfortunately, there is no formal systematic review of the literature that explores how maternal age affects neonatal survival in laboratory and wild systems. Such a review could be useful to survey the diversity of maternal ageing patterns, to investigate the conditions under which predictions from the evolutionary models succeed and fail and to better contextualize results from new studies.

Moorad and Nussey's evolution models (2016) predict that maternal effect senescence is unavoidable, but these assume the presence of age-specific genetic variation for maternal effects. Mixed evidence for the existence of maternal effect senescence across species may be expected if they vary in the degree to which their maternal genetic effects are age-dependent. Further research should focus on measuring genetic correlations between age-specific maternal effects to see whether these maternal effects are actually age-independent. However, it is important for more studies to quantify maternal age-effects more carefully. More cross-fostering experiments that control for variation in reproductive history and that take selective disappearance into account can provide the clearest estimates of these effects, whilst correctly assigning them to pre- and postnatal causes.

The failure to detect clear age-related increases in maternal contributions to offspring survival requires explanation as well, especially as previous research in another *Nicrophorus* species, *N. orbicollis,* has shown evidence for age-related increases in reproductive allocation to their offspring (Creighton *et al.* 2009; Billman *et al.* 2014). In fact, whilst Charlesworth and Léon's formal model of reproductive effort (1976) predicts increasing adaptive optima with increased age, they do not make strong predictions regarding total reproductive investment at late-ages, where selection for

total reproduction is weakest (Hamilton 1966). As reproductive effort is a proportional measure of allocation, the models actually predict total reproductive investment change with age to be represented by ever-larger fractional shares of an ever-shrinking pool of resources. Following this logic, Charlesworth and Léon (1976, p.456) are circumspect about applying their model to make inferences about reproduction effort in the very old and conclude that, "genes affecting later life are under relatively weak selective control, so the phenotype here may be relatively far from an evolutionary equilibrium…" Finally, it should be noted that maternal-age effects may be made too small to be detected if declines in offspring outcomes caused by maternal effect senescence are of similar magnitude to gains attributed to increasing reproductive effort with age. This scenario harmonizes with Charlesworth and Léon (1976) observation that there is antagonism between the evolution of reproductive senescence and reproductive effort by applying it to age-related maternal effect. This scenario is impossible to rule-out with our data. However, it seems very unlikely that an exact balance of antagonistic forces should exist for every trait investigated in our study.

Evolutionary theory makes no formal predictions regarding maternal effect senescence for other measured offspring traits, but one might expect patterns to follow qualitatively from predictions relating to juvenile survival. There is mixed evidence in the literature for maternal age affecting offspring performance aside from neonatal survival. Some systems show decline in offspring traits with maternal age: offspring longevity in *Philodina citrina* (Lansing 1947), birth weight in *Cervus elaphus* (Nussey *et al.* 2006), egg volume in *Diomedea exulans* (Froy *et al.* 2013) and offspring longevity and egg size in *Callosobruchus maculatus* (Fox & Dingle 1994; Lind *et al.* 2015). Other systems

show no effect of ageing: brood weight in *N. vespilloides* (Cotter *et al.* 2010), mean weight of offspring in *Nicrophorus orbicollis* (Trumbo 2009) and offspring longevity in *Drosophila melanogaster* (Yilmaz *et al.* 2008). There is a clear need for more theory to explore the evolution of maternal effect senescence in offspring traits other than neonatal survival. For example, models that clarify the conditions under which the Lansing Effect evolves would be an especially welcome addition to the literature, as this phenomenon is widely investigated (Comfort 1953; Butz & Hayden 1962; Klass 1977; Priest *et al.* 2002; Zehnder *et al.* 2007; Yilmaz *et al.* 2008). However, as is the case for maternal effect senescence manifested as variation in neonatal survival, there exists no systematic review of the diversity of this phenomenon.

Lastly, the age of the egg-producer appeared to have no effect upon the condition of the carer as reflected by weight change or residual lifespan. Specifically, larvae from older egg-producers did not adversely affect the carer, and no evidence of compensation for lower quality larvae was suggested. In some systems, age-related declines in offspring quality can often be buffered by "targeted reproductive effort", where postnatal maternal effects compensate for detrimental prenatal maternal effects (Cameron *et al.* 2000; Lock *et al.* 2007). However, this targeted effort may only occur when individuals have had previous mating experience and in systems where offspring quality declines with age (Lock *et al.* 2007). We found no evidence for the latter condition.

There are many advantages to measuring senescence in laboratory populations, but it is not clear to what degree laboratory findings fairly represent ageing in natural populations. Comparative research involving invertebrate and mammal species have shown the importance of contrasting laboratory/zoo and wild ageing rates, as the two can be extremely different (Bonduriansky & Brassil 2002; Carey *et al.* 2008; Dukas 2008;

Kawasaki *et al.* 2008; Rodríguez-Muñoz *et al.* 2010; Sherratt 2010; Tidière *et al.* 2017). One likely contribution to these differences is that laboratory conditions are relatively benign and free from physiological stressors, such as the need to locate and defend a resource (Scott 1998). In *Nicrophorus*, environmental stress in the laboratory can be increased to mimic natural conditions better by decreasing the resources available to the offspring or by introducing a competitor to the mother. The same principle may apply to resolving age-related increases in reproductive effort. For example, Creighton *et al.* (2009) found that female *N. orbicollis* females allocated more to their own body weight when placed on 20g mice than when they were placed on 30g carcasses, and females subsequently allocated fewer resources to current reproductive reproduction. Nevertheless, a follow-up analysis to explore these effects could shed light on the observed patterns seen in nature.

2.5 Conclusion

We performed an experiment designed to quantify the effects of maternal age upon offspring traits in a laboratory population of burying beetle. Including cross-fostering and virgin females into this design and incorporating age at death into our analysis allowed us unprecedented clarity in the biological interpretations of our results. Here, these results indicate a lack of effect of pre- and postnatal maternal age upon offspring outcomes. Contrary to predictions made from evolutionary theory, our results illustrate that maternal age effects do not always manifest. This highlights that current theory may be insufficient to account for the true diversity of ageing patterns relating to maternal care.

Chapter 3: Evaluating the diversity of maternal age effects upon neonatal survival across animal species

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3.1 Introduction

Senescence is commonly described as an age-related physiological deterioration of organismal function typically associated with increasing mortality risk (actuarial senescence) and decreasing fertility (reproductive senescence). Adequately replicated studies report actuarial and reproductive senescence in most species across most taxa (Bonduriansky & Brassil 2002; Descamps *et al.* 2008; Jones *et al.* 2008, 2014; Bouwhuis *et al.* 2009; Waugh *et al.* 2015), with especially well documented senescent declines in natural populations of wild vertebrates (Gaillard *et al.* 1994; Nussey *et al.* 2008a, 2011; Lemaître & Gaillard 2017) and laboratory invertebrates (Rose 1984; Kenyon *et al.* 1993; Bonduriansky *et al.* 2008; Galliot 2012). However, a form of ageing distinct from these manifestations of senescence has also received much recent interest: maternal effect senescence is the detrimental result of a mother's increasing age on traits associated with an offspring's' life history or fitness, such as survival, size, growth, and lifespan (Bouwhuis *et al.* 2015; Bitton & Dawson 2017; Clark *et al.* 2017; Lemaître & Gaillard 2017; Lippens *et al.* 2017). Whilst these maternal age effects are attracting increased attention, their distributions across the tree-of-life remain poorly described (Bloch Qazi *et al.* 2017). Thoroughly investigating the prevalence and degree to which these maternal age effects occur will serve to advance our current understanding of trait senescence.

As neonatal survival is profoundly important to longevity and fitness (Crow 1958; Hamilton 1966), this is an obvious focus for demographic and evolutionary exploration of maternal age effects. Demographic models have not yet been applied to data to analyse this phenomenon, but past work has aimed to interpret the biological causes of

actuarial senescence (age-related increases in mortality) by fitting mathematical models to mortality data (Ricklefs & Scheuerlein 2002). The most prominent of such functions used to describe actuarial senescence are the Gompertz, Gompertz-Makeham and Weibull Models (Gompertz 1825; Makeham 1860; Weibull 1951). The Gompertz Model imagines that age-related increases in mortality result from an exponential increase in vulnerability to sources of mortality extrinsic to the organisms. The Gompertz-Makeham Model generalizes this to include an additional parameter to account for sources of age-independent mortality. The Weibull Model views ageing as result of catastrophic intrinsic failure which increases in probability with age and assumes that age-specific causes of death are distinctive, independent and cumulative (Ricklefs & Scheuerlein 2002). Whilst it is debatable whether model fitting can by itself provide insights into the proximate biological causes of ageing, these classical demographic models do provide a convenient method for quantifying ageing rates (Pletcher 1999) especially for the purpose of comparative study (Bronikowski *et al.* 2002, 2011; Sherratt *et al.* 2011). There is no obvious reason for why these same principles cannot be applied to describe age-related maternal effects on neonatal survival.

Several hundreds of models have been proposed to elucidate the proximate causes of ageing (Medvedev 1990), including errors in protein translation, accumulation of free radicals causing cellular damage, damage from heavy metal ions to activation of ageing accelerating mutations, and age–related changes in RNA processing (Harman 1956; Orgel 1970; Eichhorn *et al.* 1979; Medvedev 1986). In contrast, there are few evolutionary models of senescence, and all share the central tenant that senescence is caused ultimately by age-related declines in the efficacy of natural selection (Hamilton 1966). Mutation accumulation (Medawar 1952) and antagonistic pleiotropy (Williams

1957) are evolutionary models that differ in details relating to how genetic architecture constrains the response to selection on age-specific traits. Population genetic models use estimates of vital rates (age-specific survival and reproduction rates) and various assumptions related to gene action to predict patterns of actuarial senescence (e.g Hughes & Charlesworth 1994), and in particular, population genetic models of mutation accumulation predict Gompertz mortality in adults (Charlesworth 2001). More recently, Moorad and Nussey (2016) applied this approach to quantify how age changes the strength of selection for age-specific maternal effects and to show how these changes cause maternal effects upon neonatal survival to evolve. They predicted that evolved demographic patterns of this manifestation of senescence are qualitatively different from actuarial or reproductive senescence. These differences include possible improvements in neonatal survival with early-life maternal ageing and faster-than-Gompertz declines in neonatal survival with late-life maternal ageing. Furthermore, this evolutionary model ascribes clear and meaningful biological causation to maternal age trajectories in the form of age-related changes in the strength of natural selection. In contrast, the classical demographic models lack clear biological cause.

Moorad and Nussey's model (hereafter referred to as the Evolutionary Model) derive selection gradients using information relating to demographic structure (age-specific rates of survival and fertility). For this reason, model predictions can be expected to be valid only when populations are near demographic and evolutionary equilibria. As classical demographic models tend not to be justified by evolutionary arguments, we expect that the performance of these models to be relatively insensitive to departures from these equilibria. It is reasonable to expect that natural populations are closer to these conditions than laboratory populations. For these reasons, one test for the

predictive value of the Evolutionary Model is to compare its goodness-of-fit to those of classical demographic models and determine if its relative performance improves when fit to natural populations.

In this paper, we address conspicuous gaps in our understanding of maternal effect senescence by performing an extensive systematic review of the literature using meta-analytical methodology. We have chosen neonatal survival as our focus for several reasons: 1) this trait's relationship to fitness is profound and well-understood conceptually (Hamilton 1966); 2) evolutionary theory explicitly models age-specific maternal effects on this trait (Moorad & Nussey 2016); 3) conventional demographic models of actuarial senescence can be adapted to describe maternal-age trajectories; and 4) associations between the trait and maternal age are observed with sufficient frequency to enable meta-analyses. This study asks two sets of questions about the nature of maternal effect senescence as it manifests on neonatal survival rates:

1. Does maternal age tend to affect neonatal survival in the majority of species across different taxa? Do these effects of age tend to be negative? What features of specific studies appear to predict effect sizes?
2. How well does the Evolutionary Model perform relative to classical demographic models? Does this performance improve in studies of natural population, as we would expect from evolutionary theory?

We find that maternal age effects are widespread across animal species, but maternal effect senescence is a general and important phenomenon in only some groups. The reasons for this variation are as yet unknown and represent an ecological and evolutionary puzzle. However, our demographic analyses provide evidence that natural

selection is a causal determinant of this manifestation of ageing, and this represents an important first step to increase our understanding of maternal-age effect variation across species.

3.2 Materials and Methods

This meta-analysis followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses ("PRISMA") guidelines (Moher *et al.* 2009) (see Fig. 3.1). A literature search was conducted in July 2017 using the online databases Web of Science and Scopus. Google Scholar was also used, but it failed to produce any papers that were not already duplicated from other databases. Search terms are provided in Supplementary Table S3.1.

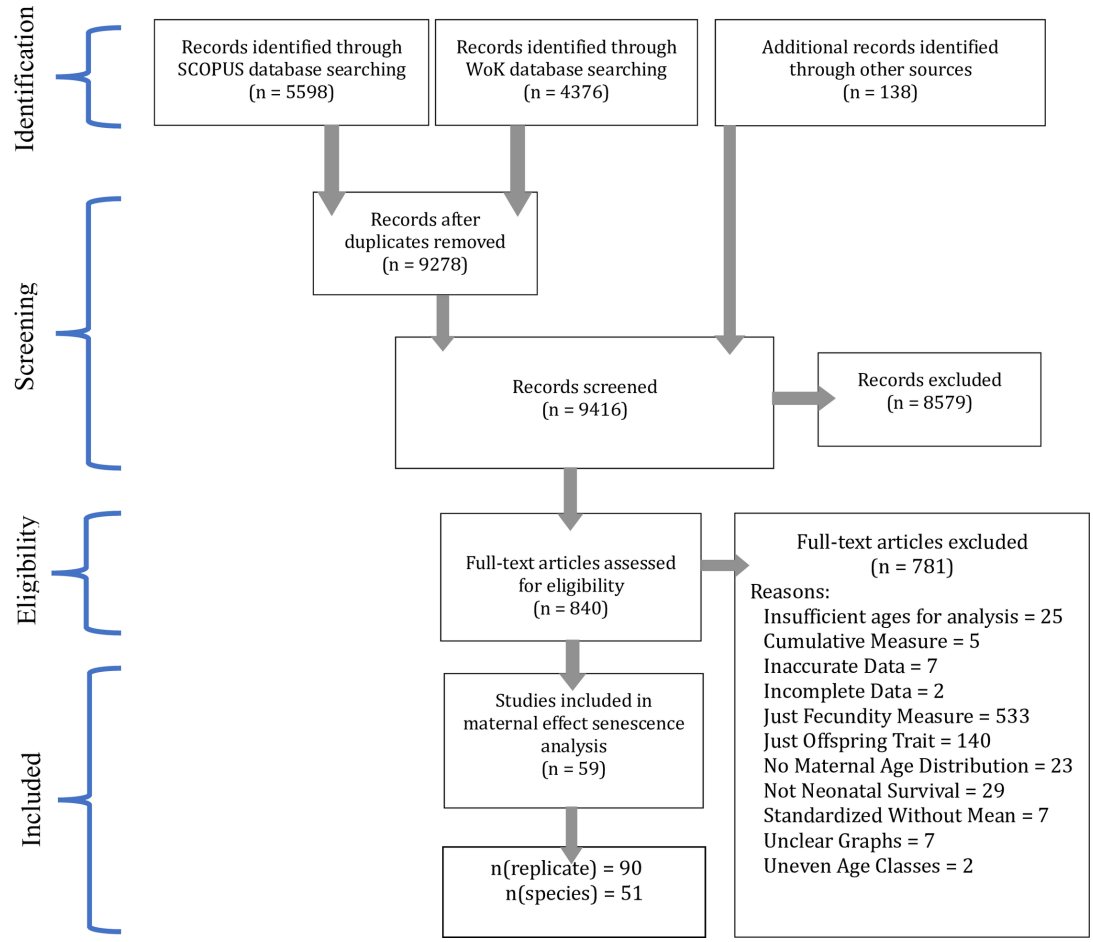

Fig. 3.1 PRISMA flow chart depicting the process and outcome of the literature search.

Accepted papers included the number of surviving and dying neonates as functions of maternal age (see Fig. 3.1). Papers were rejected if they:

1. had a title or abstract that indicated no appropriate information, or they did not contain data in graphical or tabular forms;
2. couldn't be accessed;
3. did not contain both fecundity and neonatal survival;
4. focused on humans or highly eusocial animals (as these all have highly complex social systems in which appreciable neonatal care is provided by non-maternal kin);

5. described neonatal survival solely as a function of paternal age; or
6. they included age classes with irregular intervals.

Data were extracted from accepted studies by transcription or by extraction using "WebPlotDigitizer" (Rohatgi 2014), a Google Chrome application that enabled marking of graphical axes, plotting of data points, and conversion to a replicate-specific data file. From each source, we extracted or calculated the following:

1. the number of neonates present at each maternal age class
2. neonatal survival probability at each age class;
3. female age-specific fecundity;
4. cumulative female survival rate;
5. total number of mothers; and
6. the realized maternal probability distribution (i.e. the probability of being a mother at age x, calculated as $f(x)=N_{xi}/\sum N_{xi}$, with the N_{xi} notation representing the number of offspring present at age class x).

Binomial datasets were constructed for each replicate in which each standardised age class was associated with a corresponding number of surviving and dying neonates (with corresponding trait values of 1s and 0s, respectively) reconstructed from realised maternal age distribution, age-specific fecundity and neonatal survival rates extracted from the source papers.

We standardized maternal ages by replicate-specific generation time, T, to compare effect sizes across highly variable life histories. For each replicate, i, this was calculated as the average of the maternal age distribution $f(x)$, or $T_i = \sum_x xN_{xi}/\sum_x N_{xi}$. As with any definition of generation time, this measure is sensitive to the age structure and vital

rates of the population. This may cause T to change in populations where the timing of breeding is influenced by experimenters who may wish to enhance the power of a study to detect age-related effects rather than to preserve the natural distribution of maternal ages. This likely involves the exaggeration of maternal age variance, and this will tend to increase T compared to natural values. The most likely consequence would be to cause the estimated magnitudes of maternal effects in the laboratory to underestimate those that would be measured in unmanipulated populations.

Studies were identified as belonging to Group N if data came from studies of natural populations, to Group C if data came from semi-captive populations (i.e. where there was evidence of human intervention in the form of predator exclusion or veterinary intervention), or to Group L if data came from laboratory populations. No species was studied in more than one of these contexts. Classifying studies as describing laboratory and natural populations also effectively separated species into groups with highly disparate phylogeny (Fig. 3.2) and life histories: bird and mammal species were studied in nature, are long-lived, and provide obvious maternal care; and invertebrate species were studied in the laboratory, are short-lived, and demonstrate little or no conspicuous maternal care. Semi-captive species included vertebrate mammals, birds and reptiles; all provide conspicuous maternal care. More than one binomial datasets were extracted for each species that was studied in different replicates within the same study or in multiple studies. We treated all within-species replicates as independent.

Phylogenetic trees were created using the National Centre for Biotechnology Information Taxonomy database (Federhen 2011) (to check taxonomic names for all species) and PhyloT (which converted the list of taxonomic species names into a

phylogenetic tree) (Letunic 2011), and visualised using 'ggplot2' and 'ggtree' (Wickham 2009; Yu *et al.* 2017).

The potential for publication bias should be considered in meta-analyses and tested for statistical significance whenever possible (Egger *et al.* 1997). However, statistical tests were not applicable in this study because those publications that reported maternal age effects quantified these using highly variable methods. For example, some used binomial generalised linear mixed models to report effect size estimates (e.g. Hayward *et al.* 2015) whilst other used non-parametric testing with randomisation techniques (e.g. Espie *et al.* 2000). Some corrected for selective disappearance (e.g. Potti *et al.* 2013; Hayward *et al.* 2015), whilst others did not (e.g. Rockwell *et al.* 1993; Gagliardi *et al.* 2007). Quadratic functions of maternal age were fit in some cases (e.g. Newton & Rothery 2002; Blas *et al.* 2009; Oro *et al.* 2014); linear functions were fit in others (e.g. Pugesek & Diem 1983; Rockwell *et al.* 1993). Finally, some studies investigated maternal age effects as only one of many effects of interest (e.g. Baniameri *et al.* 2005; Jha *et al.* 2012, 2014), and it may be that publication bias is less likely in these cases as multiple comparisons will increase the likelihood of detecting significant effects.

3.2.1 Does maternal age affect neonatal survival?

We estimated the effect that maternal age had on the proportion of surviving neonates for each replicate independently. We fit generalised linear models (GLMs) of neonatal survival (P) with binomial error (e) distribution and "probit" link functions to: [1] age-independent, [2] linear and [3] quadratic models of maternal age (x):

$$P(x) = A + e \quad [1]$$

$$P(x) = A + Bx + e \quad [2]$$

$$P(x) = A + Bx + Cx^2 + e \quad [3]$$

Replicate-specific log-likelihoods for all models were noted along with estimates of effect sizes and associated standard errors. We calculated Akaike Information Criterion values (AIC) for each replicate i, and model j using $AIC_{ij} = 2k_j - 2loglik_i$, where k_j is the number of parameters (one, two or three, depending upon the model – see Table 1). From these, sample-size corrected AIC values (AICc) were calculated using the formula $AICc_{ij} = \frac{AIC_{ij}+2k_j(k_j+1)}{(n_i-k_j-1)}$, where $\mathrm{n_i}$ was the number of observations for each replicate (Hurvich & Tsai 1989).

3.2.2 Do maternal age effects tend to be directional?

We used the "boot" package in R Version 3.3.3 (Kushary *et al.* 2000; R Core Team 2016; Canty & Ripley 2017) to calculate the weighted bootstrapped means of maternal age effects estimated from Models 2 (linear) and 3 (linear and quadratic) over all replicates within each species groups (n = 10,000 replicates). Weightings were made by the inverse of the estimated standard errors. Differences between L and N groups were also estimated by weighted bootstrapping.

3.2.3 Fitting demographic models

Three classical demographic models (Gompertz, Gompertz-Makeham, and Weibull) and a demographic model derived from the Evolutionary Model of maternal effect

senescence (Moorad & Nussey 2016) were fit to each replicate (Table 3.1). All three classical demographic models are intended to describe age-related increases in mortality risk, and these are not sensibly applied to situations where risk declines with age (i.e., increasing neonatal survival with advancing maternal age). The Evolutionary Model allows some initial decline in mortality risk early in life, but it is constrained to predict senescence whenever the maternal age distribution $f(x)$ decreases with increased age x. For every model, we constrained parameters accordingly (see Table S3.2). Note that Gompertz, Gompertz-Makeham, and Weibull models will converge upon age-independent solutions when neonatal mortality tends to decrease with increasing age, and the Evolutionary Model will converge upon an $f(x)$-independent solution when neonatal mortality tends to decrease as selection against neonatal survival decreases. All models were fit as optimisation functions with binomial distributions using the "optimx" package v. 2013.8.7 (Nash & Varadhan 2011) and the "Bound Optimization BY Quadratic Approximation" (BOBYQA) method from the "minqa" package v. 1.2.4 (Powell 2009; Bates *et al.* 2014) and then optimised over two steps in order to increase our confidence that our maximum likelihood solution was evaluated using starting values sampled from a broad range of biologically realistic parameter space:

Step 1: For each of the 90 replicates, 101 models of each demographic model were fit with starting values for intercepts ranging from -1 to 0 (representing neonatal survival that ranged from 0 to 100%) by intervals of 0.01. All other starting parameters were set at 0 or 1 as appropriate. This yielded 9090 solutions for each replicate-by-demographic model family combination. These were then filtered to only include parameter estimates

that provided the greatest identified log-likelihood to be used in the next step of model fitting.

Step 2: For each of the 90 replicates, 90 second optimisations were performed using all solutions derived from step 1 as starting conditions. As a consequence of this scheme, initial parameter space for each replicate-by-demographic model family analysis was sampled using reasonable parameter estimates from all replicates. The set of parameters corresponding to the model with the greatest likelihood was judged to be the maximum likelihood solution.

AICc values were estimated using each replicate-by-demographic model log-likelihoods, sample sizes and number of parameters. Calculated AICc values were used to calculate ΔAICc differences and medians between the demographic (Gompertz, Gompertz-Makeham and Weibull) and Evolutionary Models in order to assess overall performance. A different comparative perspective reduced replicate-specific AICc values to a vector of ranks for each model. For example, the model with the lowest AICc is awarded a '1', the model with the second lowest AICc gets a '2', etc. Ranks are summed over all replicates within a species and a new vector of ranks is created from the sum of the component vectors (e.g., the model with the lowest sum of ranks gets a '1'). Finally, species-specific rank vectors are summed in the same fashion to obtain species group-specific ranks.

Table 3.1 Demographic models

Demographic Model	Survival Function	k
Gompertz	$P(x) = exp\big(-exp(A + Bx)\big)$	2
Gompertz-Makeham	$P(x) = Cexp\big(-exp(A + Bx)\big)$	3
Weibull	$P(x) = \gamma exp\big(-\alpha x^{\beta}\big)$	3
Evolutionary	$P(x) = \gamma exp(-\alpha f(x)^{-1})$	2

Note - The Evolutionary Model predicts neonatal survival based directly upon the reciprocal of the probability distribution function of maternal ages, $f(x)$ (Moorad & Nussey 2016). For convenience, the Evolutionary Model was fit as a Weibull Model but with β constrained to be -1 and $f(x)$ substituted for x, but it should be emphasized that this is not a special form of the Weibull function.

3.3 Results

59 papers met our search criteria. Of these, seven provided data from semi-captive populations, 26 provided data from laboratory populations and 26 derived from natural studies. Some papers included replicate populations (e.g., multiple strains or different environmental conditions for a single species). In total, 90 datasets were extracted and analysed (see Table S3.3). These replicates represented 20 invertebrate, 13 mammal, 17 bird, and one reptile species. A preliminary search of plant literature was also conducted, however due to low numbers of acceptable papers, we focused our analysis solely on animal species.

3.3.1 How does maternal age affect neonatal survival?

Replicate-specific results from the GLMs are given in Table S3.4. As indicated by comparisons of AICc values, the age-independent models were best in 7 cases, linear

age effect models were best in 18 cases, and quadratic age effect models were best in 65 cases (out of a total of 90 replicates). Summing AICc values over all replicates indicated a strong preference for the quadratic model of maternal age on neonatal survival (ΔAICc Age-Independent: -81920; ΔAICc Linear: -6721). 69 of the 90 measured offspring outcomes had negative quadratic effects. The weighted bootstrapped means of the quadratic effects were statistically negative when pooled over all species (mean = -0.197, bias corrected 95%-tiles = -0.321, -0.113) and within each group: mean(N) = -0.144 (-0.246, -0.090); mean(L) = -0.212 (-0.414, -0.088); and mean(C) = -0.504 (-1.195, -0.197). The bootstrapped mean difference between L and N suggested that these two groups were not statistically different (mean difference = -0.068, 95%-tiles = -0.109, 0.245). However, the strong tendency across all species towards negative quadratic effects of age indicates that linear models of maternal age tend to underestimate maternal effect senescence experienced by older females (or overestimate maternal effect improvement in the old). In light of this finding, we re-focused our question to evaluate the linear effects of maternal age on old females only, where old defines ages greater than T (i.e., the mothers that are older than average). See Fig. S3.1 for the among-replicate distribution of oldest mothers surveyed.

The distribution of maternal age-effects in old mothers is illustrated in Fig. 3.2. The mean effect of maternal ages was statistically negative over all species pooled together (mean = -0.452, bias corrected 95%-tiles = -0.621, -0.301), over species from Group L (mean = -0.671, bias corrected 95%-tiles = -0.908, -0.456) and over species from Group C (mean = -0.366, bias corrected 95%-tiles = -0.986, -0.073). Whilst the estimated mean effect within Group N was also negative, it was not statistically different from zero (mean = -0.062, bias corrected 95%-tiles = -0.1374, 0.028). As the distribution of effect sizes

shown in Fig. 3.2 suggested a profound difference between birds and mammals, we separated Group N into new sub-groups (N_B for natural bird studies and N_M for natural mammalian studies). In order to test for an overall difference between Groups L, N_B, and N_M, we applied a non-parametric Kruskal-Wallis test (N.B. Group C species were removed from this analysis as they were few in number, contained both mammalian and bird species, as well as a reptile, and they exhibited a range of human interventions). We found a significant effect of species grouping on measured late-age effect sizes ($\chi^2(2) = 18.399$, $p < 0.001$). A Pairwise Test For Multiple Comparisons of Mean Rank Sums (Nemenyi-Tests) from the PMCMR v4.3 package (Pohlert 2014) indicated significant differences between Groups N_B and N_M (Tukey HSD = 4.625, $p = 0.003$) and between Groups N_B and L (Tukey HSD = 5.415, $p < 0.001$) but not between Groups N_M and L (Tukey HSD = 1.301, $p = 0.628$). Overall, these results strongly suggest that late-age maternal effects in laboratory invertebrates and wild mammals (N_M mean = -0.578, bias corrected 95%-tiles = -0.699, -0.485), are stronger than in natural populations of birds, where the mean effect over all such studies appears to be absent (N_B mean = -0.007, bias corrected 95%-tiles =-0.086, 0.085). Note that these effect sizes are scaled as survival fraction changes per generation (e.g., the mean effect pooled over all studies is a 45.2% decrease in survival rates for a +T change in maternal age).

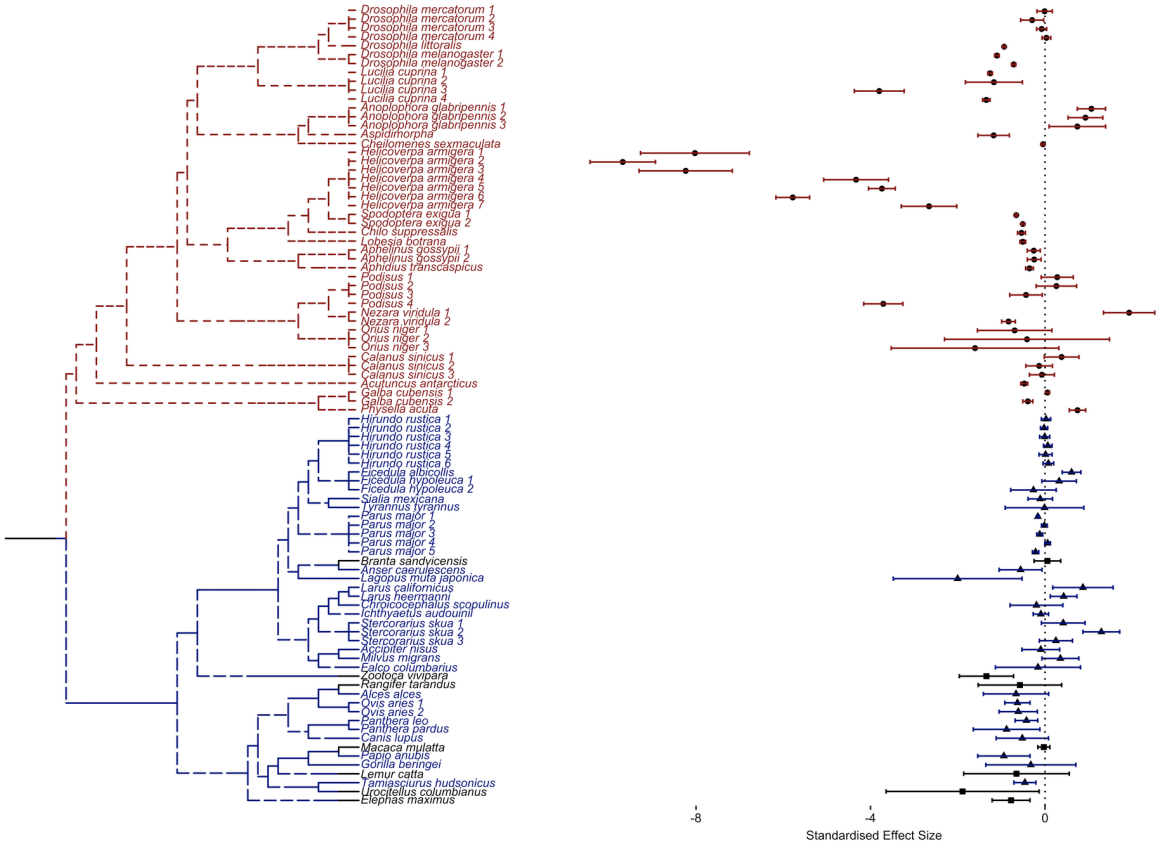

Fig. 3.2 Phylogenetic tree of species included in the comparative analysis with accompanying replicate-specific effect sizes for old maternal age classes (age greater than *T*).

Note - The blue text indicates those species evaluated in nature, the red text indicate those assessed in the laboratory, and the black text represents species from semi-captive populations. The forest plot shows the maternal age effect (standardized by generation time, *T*) on neonatal survival across all replicates. Circular points represent effect size estimates for laboratory species, triangular points represent those for natural species and square points represent those for semi-captive species. Error bars around the estimate represent 95% confidence intervals.

3.3.2 How well do demographic models fit?

We compared the fits of various demographic models of neonatal survival to extracted data from a variety of animal species in natural and laboratory populations (see Tables S3.5-6). As assessed by median ΔAICc values, the Evolutionary Model performed worse than all three of the classical demographic models (Gompertz: +41.4, Gompertz-Makeham: +26.1, Weibull: +43.9) in laboratory populations (Fig. 3.3A-C). These performance rankings persisted when replicate-specific AICc comparisons were condensed into species-specific model rankings, and ranks were weighted and summed as described above (Table 3.2).

By comparison of ΔAICc values, the Evolutionary Model appeared to have performed better than the classical demographic models (Fig. 3.3A-C) in natural populations. The median ΔAICc between the Evolutionary and Gompertz Models was -0.242. In 23 cases, the best fit Gompertz and Evolutionary Models both converged on age- and selection-independent solutions with identical log-likelihoods. This led to identical ΔAICc measures as both types of models share the same number of parameters (two). These non-informative ΔAICcs were removed from the calculation of the median. Gompertz-Makeham and Weibull Models were less favoured in these situations because they fit three parameters; these were included in calculations of these median differences. From median ΔAICc value, the Evolutionary Model outperformed Gompertz-Makeham (-0.941) and Weibull Models (-1.003). When species-specific model ranks were compared rather than median ΔAICc values, the Evolutionary Model performed best (Table 3.2).

In both comparisons of ΔAICc values and species-specific models ranks in semi-captive populations, the Evolutionary Model was found to outperform the demographic models

when assessed by median ΔAICc (Gompertz = -3.565, Gompertz-Makeham = -0.504, Weibull = -1.010) and by summed ranks (Fig. 3.3A-C and Table 3.2).

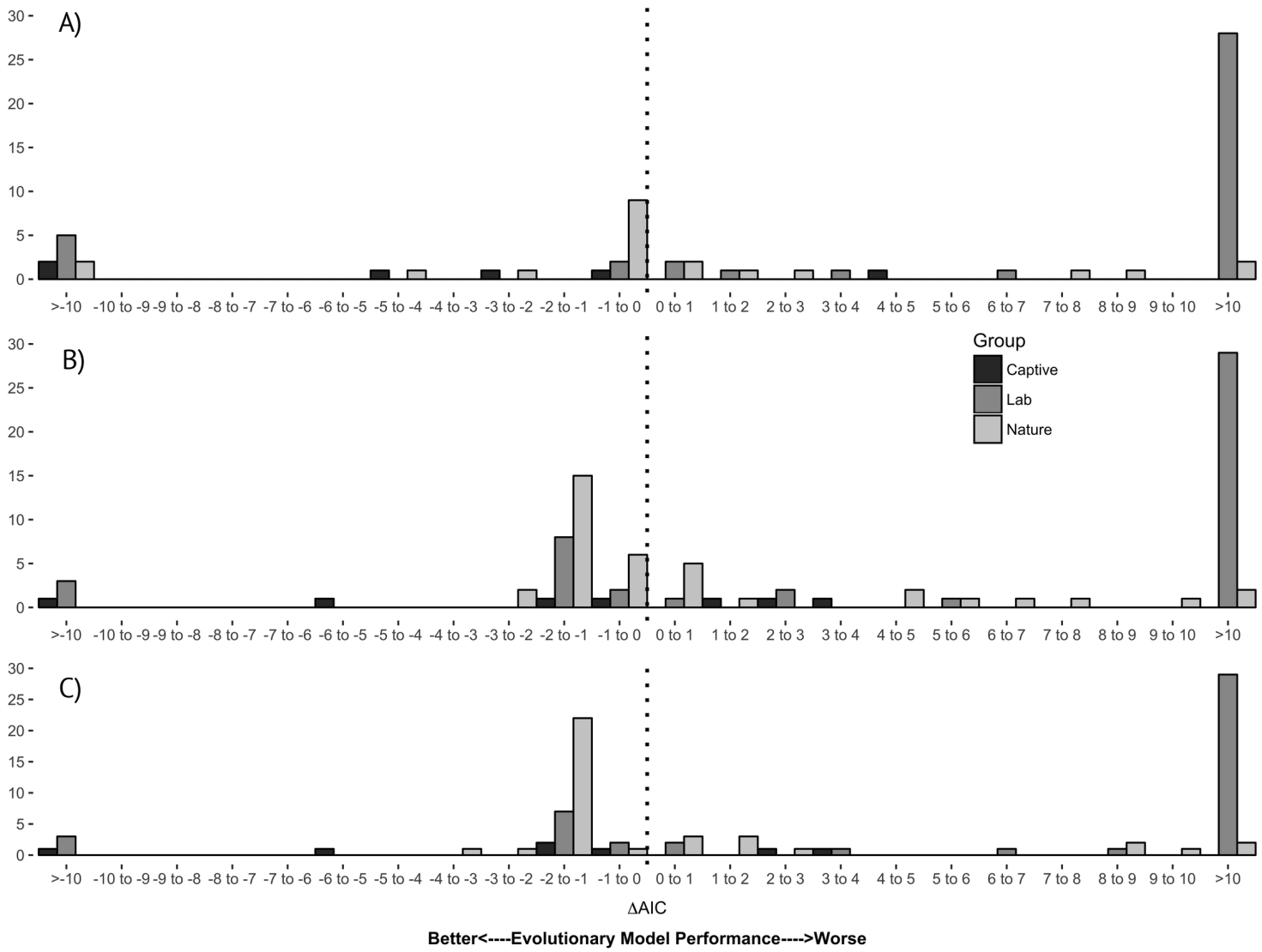

Fig. 3.3 Histograms representing ΔAICc differences between the Evolutionary Model and the A) Gompertz, B) Gompertz-Makeham and C) Weibull models.

Note - Bars in black represent counts from captive replicates, dark grey from laboratory replicates and light grey from natural replicates. On 23 occasions, the ΔAICc between the Gompertz and Evolutionary Models was 0; these were omitted from the figure.

Table 3.2 Species group-specific ranking of the four models in Groups L, N and C.

Ranking	Gompertz			G-Makeham			Weibull			Evolutionary		
	L	C	N	L	C	N	L	C	N	L	C	N
1st	5	1.5	5.5	1	1	8	13	1	2.5	1	3.5	8
2nd	8.5	1.5	9.5	7.5	2	1	2	2	7	2	1.5	6.5
3rd	4.5	2	5	8.5	4	9.5	3	0	4	4	1	5.5
4th	2	2	4	3	0	5.5	2	4	10.5	13	1	4
Total	43.5	18.5	55.5	53.5	17	60.5	34	21	70.5	69	13.5	53.5
Overall Rank	2	3	2	3	2	3	1	4	4	4	1	1

Note - Ranked from best (1st) to worst (4th) based on how predictive the four models were when comparing AICcs. Lower ranks indicate better models. Scores with 0.5s represent situations in which a tie occurred; an average was taken in these situations.

3.4 Discussion

3.4.1 Maternal age effects

Maternal age appeared to affect neonatal survival in 83 of 90 studies accessed in this review (91%), and these effects appeared to be widespread across divergent taxa, life histories and environments. Whilst these results argue persuasively that maternal age effects in late-life are of general importance, phylogenetic constraints may be important in determining whether these effects are directional: increased maternal age clearly tends to become progressively more deleterious in laboratory invertebrates, semi-captive vertebrates, and mammals in nature, but there is no statistical support for widespread late-age maternal senescence in natural populations of bird species. Laboratory populations of invertebrates appeared to experience insignificantly faster

maternal-age-related declines in neonatal survival than wild mammals at late ages (67.1% vs 57.8% decline per unit of generation time), but it's possible that this difference is an underestimate owing to bias associated with estimations of generation time made from experimental studies (see Materials and Methods).

On the other hand, there is a clear and dramatic difference between senescence rates between wild mammals and wild birds (57.8% vs 0.7% decline per unit of generation time). This study lacks the means to explain the causes of this difference, but because both sorts of animals provide prenatal and postnatal care we can reason that it cannot be explained by qualitative differences in the types of maternal care provided. Beyond this, we can only speculate how differences in phylogeny or attendant life-history patterns might generate this variation. One possibility could be that mammalian maternal care is more dependent upon physiological condition than avian maternal care, and this condition degrades with increased maternal age. This borrows from suggestions made in the evolutionary literature that interactions may exist between age-related physiological degradation and condition-dependent environmental hazard that affect age-specific mortality (Williams & Day 2003); this model has been used to suggest that flight reduces that environmental hazard, and this may help explain the oft-made observation that birds live longer than mammals (Williams 1957; Holmes & Austad 1995). As nesting in arboreal or other sites that are protected from predators (such as cliffside or offshore rocks) is often made possible by flight, it could be that flight insulates neonatal birds from effects caused by the physiological senescence of their mothers. Comparisons of maternal age effects on neonatal survival measured in captivity (where the physiological effects of ageing can be suppressed) and in the wild (where

they are not) in both bird and mammal species could be made to evaluate this suggestion.

Selective disappearance might explain the dramatic differences between maternal senescence rates in birds and mammals if female survival and maternal quality were more closely associated in birds than in mammals. If so, then female deaths leading up to later ages would cause the preferential removal of poor mothers before neonatal survival could be measured in the post-selection cohort late-in-life; this would lead to a situation in which cohort-level measures of ageing underestimate the true degree of senescence experienced by individuals. Selective disappearance has been discussed at length in the context of actuarial (Vaupel *et al.* 1979; Vaupel & Yashin 1985), reproductive (Bouwhuis *et al.* 2009) and physiological senescence (Nussey *et al.* 2011), but it has only recently been explored in the context of maternal effect senescence (Ivimey-Cook & Moorad 2018a). An effect of selective disappearance upon neonatal survival been detected in two studies of seabirds (van de Pol & Verhulst 2006; Zhang *et al.* 2015) but not in a wild population of Soay sheep (Hayward *et al.* 2013) or in a laboratory study of a beetle with conspicuous maternal care (Ivimey-Cook & Moorad 2018a), but we lack a biological model that might explain why the phenomenon should be more important in birds than in mammals or invertebrates. Nevertheless, this possibility could be easily evaluated if more future studies of maternal effect senescence report and correct for these effects. This is a simple addition to observational or experimental analyses, requiring only the fitting of time-of-death into standard statistical models, and it should be standard practice for all measurements of maternal effect senescence whenever possible. Finally, we note that the evolution of maternal effect senescence requires an amenable genetic architecture, but, we lack a reasonable

biological model that might predict why maternal effect genes in invertebrates and mammals should be more similar in this respect than birds. Perhaps future quantitative genetic analyses (see below) applied to mammal and bird species could shed some light on this possibility.

3.4.2 Demographic comparisons

Classical demographic models treat age as a predictor of mortality. In contrast, the Evolutionary Model uses age-specific selection, which is derived from age-specific survival and fertility, as a predictor. Both comparative measures used in this study (summed ranks and median ΔAICc values), identified the Evolutionary Model as superior to the classical demographic models when fit to natural populations. One obvious interpretation is that an age-related relaxation in the strength of selection is a causal determinant of maternal effect senescence, and this manifestation of ageing has an evolutionary component. Added support for this interpretation comes from the relatively poor performance of the Evolutionary Model in laboratory populations, where estimates of current selection should correspond poorly to the long-term intensities of age-specific selection for maternal effects on neonatal survival. However, we cannot ignore the fact that the two environments considered here are not randomly distributed across the tree-of-life; the species represented in wild animal studies are very different from those studies in the laboratory. It is possible that evolution by natural selection has shaped maternal age effects in birds and mammals more than it has caused these effects in invertebrates, but it is difficult to imagine how that difference might have arisen, and this explanation requires an effective, but yet-to-be proposed, non-evolutionary

mechanism to explain maternal effect senescence in invertebrates. We hope for future work on this subject, such as more studies of maternal effect ageing in insect species with postnatal maternal care (e.g. Ivimey-Cook & Moorad 2018a) and observations of senescence in single species assessed in both laboratory and natural conditions (Kawasaki *et al.* 2008).

As with the classical evolutionary theory of senescence (Williams 1957; Hamilton 1966; Charlesworth 1994), the evolutionary model of maternal effect senescence demonstrates that age-attenuated selection is inevitable late-in-life (Moorad & Nussey 2016). However, natural selection can shape evolution only to the degree made available by the underlying genetic architecture (Lande 1979). In the context of maternal effect senescence, this means that: the genetic causes of maternal effects on neonatal survival must be age-dependent to some degree and the ranked-order of these genetic effects must change with maternal age. Direct estimates of maternal genetic effects on neonatal effects in a wild population of red deer (Nussey *et al.* 2008b) provide some evidence that this first condition is met by observing an age-related increase in genetic effects for maternal contributions to offspring birth rate (a predictor of survival). To our knowledge, however, the second evolutionary condition has yet to be tested in the wild. Doing so would involve the measurement of genetic correlations for age-specific maternal contributions across ages and testing for correlations that can be significantly bounded away from +1. Such tests should be applied to confirm or refute directly the existence of age-dependent maternal effects on neonatal survival that are inferred by this study.

Finally, it should be emphasized that future conceptual advancements in evolutionary theory could provide better models to explain maternal effect senescence, perhaps by embellishing upon the relatively simple population genetic model of Moorad and Nussey

(2016). There are many features known to be important to reproductive and actuarial senescence that are not included in this model, such as: across-age genetic pleiotropy (Williams 1957; Charlesworth 2001), selective disappearance (Vaupel *et al.* 1979; Vaupel & Yashin 1985; van de Pol & Verhulst 2006), mutational bias (Moorad & Promislow 2008), density- and condition-dependent effects (Abrams 1993; Williams & Day 2003), and within-age trade-offs (Charlesworth & Leon 1976). In addition to these, cross-generational life history trade-offs or other genetic pleiotropy (e.g. Hadfield 2012) could be important to the evolution of maternal effects. Any or all of these can contribute to extant patterns of maternal ageing.

3.5 Conclusion

This study provides the first comprehensive and comparative assessment of maternal age effects on neonatal survival across several diverse animal species. The first goal was to survey across 51 animal species in 59 published papers for interesting distributions of effect sizes; we found that maternal age tends to be an important determinant of neonatal survival across multiple animal taxa. Furthermore, we found that these maternal age effects tended to worsen over time in laboratory invertebrate and wild mammal populations. Surprisingly, this strong signal of senescence was lacking in wild populations of birds. This profound divergence represents a puzzle that deserves future attention. The second goal was to assess these patterns from an evolutionary perspective and to gauge whether natural selection could explain extant patterns of maternal effect senescence. Comparing goodness-of-fits from relevant evolutionary models of senescence to those from demographic models of mortality revealed that the

strength of age-specific natural selection was superior to age as a predictor of ageing patterns. Taken together, these findings indicate that maternal age effects upon a trait of fundamental ecological, evolutionary, and demographic importance are widespread and were likely shaped by evolutionary forces.

Chapter 4: What can natural selection tell us about diversity of ageing patterns across species?

4.1 Introduction

Many comparative studies have attempted to describe the diversity in ageing patterns across animal species (Promislow 1991; Gaillard *et al.* 1994; Ricklefs & Scheuerlein 2001; Ricklefs *et al.* 2003; Jones *et al.* 2008, 2014; Péron *et al.* 2010; Nussey *et al.* 2013; Lemaître & Gaillard 2017; Ivimey-Cook & Moorad 2018b). In particular, recent work by Jones *et al.* (2008) compared the rates of senescence amongst 19 species of birds and mammals. They found widespread evidence for senescence in the wild, with birds showing a lower rate of senescence in survival and recruitment than mammals. More recently, Jones *et al.* (2014) used life tables to analyse patterns of mortality and reproduction across a multitude of taxonomic groups, including 11 mammalian species, 12 other vertebrates, 10 invertebrates and 12 plant species. For both mortality and fertility, there was large variation amongst the patterns of senescence across taxonomic groups, with observable increasing, decreasing, constant, convex, and concave-shaped age-trajectories of measured vital rates (Jones *et al.* 2014). However, despite extensive comparative work investigating the diversity in ageing trajectories, the ultimate cause for this variation remains unclear.

In order to better understand the evolutionary underpinnings to this variation, one must consider theory provided by William Hamilton (1966) that defines how natural selection acting on survival and fertility declines with age. In particular, selection against mortality can only begin to decline after the onset of reproductive maturity, and selection acting on fertility is maximised at birth and declines with advancing age. Making simplifying assumptions regarding genetic architecture, Hamilton concludes that senescence was an inevitable and universal outcome of evolution and followed

from the age-related declines in selection (Hamilton 1966). Whilst Hamilton makes no insights into the genetics of senescence, his descriptions of the strength of selection on vital rates are both correct and complete (Lande 1982; Charlesworth 1994; Moorad 2014). Consequently, his predictions regarding declining selection have often been incorporated into population genetic models that make more realistic and sophisticated assumptions of gene action (See Charlesworth 1994, 2001; Baudisch 2005; Moorad & Promislow 2008).

Recent attention has focused on organisms that do not exhibit the '*pro-senescent*' decline predicted by Hamilton, and instead show negligible or even decreased mortality risk with increased age (Finch 2009) (examples from Jones *et al.* (2014) include: hydra, tortoises, coral, long-lived fish). Similar patterns of both negligible and negative senescence have been observed in various plant species (Baudisch *et al.* 2013; Caswell & Salguero-Gomez 2013). As well, these non-Hamilton-like observations or situations of negative senescence have been explored mathematically by Vaupel *et al.* (2004) and later by Baudisch (2005, 2008). In particular, they noted that whilst Hamilton proved mathematically that selection acting for fertility and against mortality must decline with increased age, there are a multitude of other senescent indicators (a measure of the force of natural selection acting on a trait, see Table 1 from Baudisch 2005) that could remain constant or even increase with age in relation to the shape of age-specific mortality and fertility (Baudisch 2005, 2008). This widespread evidence for non-Hamilton-like ageing trajectories from real populations has led to the suggestion by some that senescence is not universal and that observed variation in ageing trajectories is not predicted by classical evolutionary theory (Vaupel *et al.* 2004; Jones *et al.* 2014; Jones & Vaupel 2017). This suggestion is not without controversy however, with some

researchers questioning the validity of comparing demographic trajectories of highly laboratory-adapted species to those of natural field populations and ignoring the impact that variability in environment has on ageing rates (Gewin 2013). Furthermore, whilst Jones et *al.* (2014) describe the large variation in ageing patterns across the tree-of-life, it has been argued that this observation of diversity alone is not sufficient to refute existing evolutionary theory. For example, Gewin (2013) suggests that empirical analyses of trade-offs that occur between reproduction and mortality are required first. Similarly, the use of clonal organisms (as per Vaupel *et al.* 2004) in studies describing patterns of negligible or negative senescence has been questioned (Rose *et al.* 2007; Finch 2009; Roach 2016). More specifically, this argument was highlighted by Rose *et al.* (2007), who commented that whilst these models of negative senescence are interesting from a life history perspective, they are not applicable to tests of Hamilton-like ageing as they involve clonally reproducing organisms. An obvious direct step for understanding the evolution of variation in ageing trajectories better is to survey the distribution of departures from Hamilton-like ageing across multiple species.

Existing theory suggests several mechanisms that may cause departures from Hamilton-like predictions of vital rate senescence. These include:

1. Demographic heterogeneity or among-individual frailty, whereby the selective removal of low quality individuals promotes a reduction in a population's subsequent mortality risk and may produce observable mortality plateaus or mortality deceleration (Vaupel & Yashin 1985; Vaupel *et al.* 1998; van de Pol & Verhulst 2006; Nussey *et al.* 2011). This among-individual heterogeneity has also been shown to affect traits other than survival, such as body mass in ungulate species (Nussey *et al.* 2011), fertility traits in female

great tits (*Parus major*) (Bouwhuis *et al.* 2009) and reproductive performance in red-billed choughs (*Pyrrhocorax pyrrhocorax*) (Reid *et al.* 2003). However, research has shown that the effects of selective disappearance do not always manifest (Simons *et al.* 2016; Ivimey-Cook & Moorad 2018a) It is possible that populations that experience high levels of between-individual heterogeneity exhibit ageing patterns that appear to demonstrate negative senescence even if the mortality risk faced by individuals strictly increases with age. Thereby, in order to test for the presence of demographic heterogeneity contributing to non-Hamilton-like ageing, we used a likely correlate of selective disappearance, the degree of iteroparity, measured as the distribution of females around the mean age of reproduction (standard deviation of generation time). A large degree of iteroparity indicates high potential for demographic heterogeneity to cause fertility to demonstrate negative senescence because females will tend to realize fertility over greater ranges of ages.

2. Whilst negative genetic correlations may maintain or even strengthen senescence, for instance through pleiotropic trade-offs between increased reproductive effort in early-life and decreased survival in late-life (Williams 1957; Charlesworth & Leon 1976; Nussey *et al.* 2008b; Boonekamp *et al.* 2014), positive genetic correlations across ages can contribute to deviations from Hamilton's expectations (see Charlesworth 2001; Wachter *et al.* 2013). Particularly relevant life-history traits that are often associated with positive genetic correlations include: increasing organismal size with age, a trait especially applicable in species that show indeterminant growth where age-

related enlargement in body size typically results in reduced mortality rates and increased fertility rates (Sogard 1997; Heino & Kaitala 1999; Jones *et al.* 2014), a clear violation from predicted age-related declines. Additionally, we may also expect positive genetic correlations between extended late-age lifespan and social care if prolonged maternal survival is positively correlated with inclusive fitness benefits for females through protracted social care of offspring and grand-offspring. This reduction in late-age mortality rate, mediated through enhanced fitness via social care of progeny, is often termed the "Grandmother" (Hawkes *et al.* 1998) or "Mother" (Williams 1957) hypotheses respectively. To test for possible evidence of positive genetic correlations affecting adherence to evolutionary theory, we included adult body mass and parental care duration as life history covariates in our multivariate analysis.

Describing the distribution of deviations from Hamilton's expectations will allow us to firstly identify whether natural selection can accurately describe patterns of vital rate ageing. As well, there is a need to identify the various taxonomic groups in which deviations from model predictions are most likely to occur. Additionally, which type of ageing (actuarial vs reproductive) are best explained by predictions from simple evolutionary genetic theory as this could highlight potential differences in trait complexity and underlying genetic architecture. Lastly, what life history features are most commonly associated with departures from evolutionary theory, which may reveal potentially larger biological processes that interfere with predictions from simple evolutionary theory.

In this study, we measured population-specific correlations between observed and predicted vital rate data across 136 populations, representing 45 animal species, to quantify population-specific violations from Hamilton-like ageing. This was done with the aim to visualise and describe the distribution of violations across multiple animal species and identifying taxonomic hotspots of non-Hamilton-like ageing. Furthermore, we examined a number of key life history traits as indicators of processes that potentially distort predictions from simple evolutionary theory, specifically: length of generation time, the degree of iteroparity, adult body mass, and parental care duration. Specially, we used these indicators as we wished to know what life history constraints were readily contributing to non-conformance of observed vital rates to evolutionary theory and potentially link these to underlying biological processes.

4.2 Materials and Methods

4.2.1 Data collection

Selection for vital rates derive from vital rates (Hamilton 1966), and vital rates define actuarial and reproductive senescence (Finch *et al.* 1990). Sufficient data for describing these are given in the form of population projection matrices known as 'Leslie matrices' (Leslie 1945). These matrices represent discrete, age-structured models and contain data on age-specific fertility (along the top row) and age-specific survival (on the sub-diagonal).

We used Leslie matrices accessed from two sources. The first was the COMADRE v.2.0.1 open-access database (Salguero-Gomez *et al.* 2016a), a collection of 1927 population matrices extracted from 508 studies covering 405 taxonomically accepted

animal species. Appropriate data were selected using the "subsetDB" function from the Mage package (Salguero-Gómez *et al.* 2015; Salguero-Gomez *et al.* 2016a) in R version 3.3.3 (R Core Team 2016) and reconfigured as Leslie matrices. The matrices used in our analyses satisfied the following criteria:

1. vital rates were structured exclusively by age (e.g., no stage structure);
2. matrices had to contain data on at least three age-classes, and two of these must have been from reproductive ages;
3. The matrix could not contain NA values for vital rates;
4. The matrix could only contain values on the top row (age-specific fertility) and the sub-diagonal (age-specific survival) of the matrix;
5. vital rates must have described female, asexual or hermaphroditic individuals;
6. data must have come from natural populations; and
7. Individual and pooled matrix composites were used. An individual matrix was classified as "a population model constructed for a single study x species x population x treatment x period combination"; whilst a pooled matrix was classified as "a population model that has been constructed by pooling individual-level demographic data across populations or periods" (Definition from COMADRE User Guide, 2017). Priority was placed on using pooled matrices, however if these were not available then individual matrices were used.

Applying these criteria to data from COMADRE (Salguero-Gomez *et al.* 2016a) provided us with 115 matrices representing 26 species. The second source of data was DATLife (DATLife Database, Max-Planck Institute for Demographic Research (Germany) available at www.datlife.org), a curated database of vital rates for 277 species. 21 species were

selected for analysis as they contained data on both age-specific mortality and fertility of females from the same natural population. The species sets selected from the two databases did not overlap with one exception, *Marmota flaviventris*, a species of marmot. In this case, we treated both records as separate populations.

4.2.2 Formation of age-specific vital rates and selection gradients

For each population matrix, age-specific survival, the sub-diagonal of the Leslie matrix, was converted to age-specific mortality using, $\mu_x = -\ln(P_x)$. Age-specific fertility (m_x) was taken directly from the top row of the population matrix. Selection gradients come from Hamilton sensitivities (Hamilton 1966), these were converted to selection gradients (scaled by generation length) by multiplying by *T* (Lande 1982; Moorad 2014), where $T = \sum x l_x m_x / R_0$ (Charlesworth 1994). For clarity, l_x is cumulative survival to age *x* and m_x is age-specific reproduction, and R_0 is reproductive rate. Selection for age-specific mortality is $\beta_{\omega,\mu_x} = -\sum_{y=x+1} l_y m_y e^{-ry}$, and selection for age-specific fertility is $\beta_{\omega,m_x} = l_x e^{-rx}$. *r* is the Malthusian population growth rate, calculated as the natural logarithm of the dominant eigenvalue of the population-specific Leslie matrix.

4.2.3 Assessing the strength of associations between selection and evolutionary predictions

For each species, correlations were made between observed vital rates and vital rates predicted by two different evolutionary models of senescence. These correlations were calculated independently for age-specific mortality (ρ_μ) and age-specific fertility (ρ_m) and were weighted by cumulative survival using the "weightedCorr" function from

the wCorr package v1.9.1 (Emad & Bailey 2017). This weighting scheme is expected to best reflect the number of observations used to calculate the relevant vital rates and causes age classes with the highest number of observations (i.e., in early-life) to exert more influence over the correlation than those with fewer (i.e., in late-life).

Two population genetic models were chosen to predict evolutionary endpoints by virtue of their extremely simple, yet non-trivial, models of genetics. The first model derives from population genetic models of age-specific mortality (Charlesworth & Hughes 1996) and age-specific fertility (Charlesworth 2001); these imagine that new mutations that affect age-specific mortality and fertility do so additively on these scales. Mutations are assumed to affect either fertility or mortality (not both), and each mutation can affect these traits at only one specific age. All mutational effects are identically distributed across all ages. Quantitative model predictions depend upon both population-specific genetic and ecological parameters (e.g., per-generation mutation rates and extrinsic hazard rates) with values that are unknown in all but a very few species, but predictions sufficient for the purposes of this study are insensitive to these parameters because these are assumed to be age-independent (and therefore do not contribute to correlations). The relevant vital rate predictions that arise from these population genetic models are given below (see S4.1 for complete derivation of predicted vital rates). For mortality specific to any age x, selection is inversely proportional to the negative natural logarithm of age-specific mortality,

$$ln\big(\mu(x)\big) \sim -\frac{1}{\beta_{w,\mu_x}}.$$

For age-specific fertility, where m_0 represents fertility at the first age of reproduction, selection is inversely proportional to the senescent loss of fertility (on the proportional scale),

$$1 - \frac{m_x}{m_0} \sim \frac{1}{\beta_{w,m_x}}.$$

The second model relaxes the assumptions of additivity from the first models, but new mutational effects are still assumed to be independent and identically distributed across ages. In this case, quantitative predictions are impossible, and the evolutionary predictions revert to the expectation that survival and fertility is greatest at the ages that selection for these vital rates is highest. These qualitative predictions correspond exactly to those made by Hamilton (1966).

Weighted Pearson correlations are estimated between vital rates and predictions that derive from the population genetic models, which we hereafter refer to as 'Parametric' associations. Because the second model's predictions must be qualitatively identical to those of the population genetic models, weighted Spearman ranked correlations are made using the same data. We refer to these as 'Non-parametric' associations. Finally, for both parametric and non-parametric associations, a matrix-specific difference between associations was calculated as $\rho_\Delta = \left(\rho_\mu - \rho_m\right)/2$. The matrix-specific difference (ρ_Δ) quantified the comparative ability of both evolutionary models to predict patterns of observed age-specific fertility and mortality rates. A positive association difference indicated an improved performance in describing mortality ageing trajectories compared to fertility, whilst a negative value suggested the opposite trend. A grouping around zero suggested that the evolutionary models were showing equivalent performances, either poor or good, when describing observed

mortality and fertility rates. In total, six estimates were made for each population matrix (ρ_μ, ρ_m, and ρ_Δ for both parametric and nonparametric models). Lastly, in order to statistically investigate differences between the non-parametric and parametric correlations for both age-specific fertility and mortality, a bootstrapped Kologomorov-Smirnov test was performed (from the Matching package, Sekhon 2011) in R version 3.3.3 (R Core Team 2016) which allows for two sets of distributions to be compared for degree of similarity.

4.2.4 Identification of important life history variables

In order to better understand possible biological processes that may be leading to non-conformance between observed vital rates and predictions from simple evolutionary theory, we constructed multivariate generalised linear mixed effect models using ASReml v.4.1 (Gilmour 1997) with ρ_μ, ρ_m, and ρ_Δ (parametric and non-parametric) as response variables. The following life history components were used as predictors:

1. mean generation time, $T = \sum x l_x m_x e^{-ry}$;
2. degree of iteroparity, defined as the standard deviation of T ($\sigma_T = \sum (x - T)^2 * l_x m_x e^{-ry}$);
3. taxonomic class, consisting of mammals (species n = 19, matrix n = 99), birds (species n = 8, matrix n = 8), ray-finned fish (species n = 11, matrix = 14), reptiles (species n = 5, matrix n = 5) and one polychaete species (matrix n = 10);

and when sufficient data were available, the following were also used:

4. natural logarithm of adult body mass in kilograms (M_A); and

5. parental care duration (P_C), the sum of pre- and postnatal care (until independence) duration.

Whilst *T* and σ_T were calculated directly from the population matrix, M_A and P_C were taken from external sources such as AnAge (De MagalhÃes & Costa 2009), Fishbase (Froese & Pauly 2017), Animal Diversity Web (Myers *et al.* 2006) or the Encyclopaedia of Life (Parr *et al.* 2014). These are life history databases that collate information on key traits from published literature. For some species, data on several life history traits were unavailable. In these circumstances, data from closely rated species from the same genus was used (*n* = 3). Congeneric data has been used in this way in previous comparative analyses (see Jones *et al.* 2008). Multivariate multiple regression mixed models were then constructed with two sets of predictors.

1. The *simple model*: *T*, σ_T, and taxonomic class were fit as fixed effects using all species-specific correlations (n = 82). Correlations were removed from the analysis if they either: a) contained NAs for any vital rate or b) were the sole species in a taxonomic group (e.g. polychaetes were only represented by one species, *Nephyts incise*). Humans were excluded from the study, as available populations were assumed to be far from evolutionary equilibria (Corbett *et al.* 2018).
2. The *life history model*: All collected life history traits were fit as fixed effects in the 67 populations that included complete measures of both M_A and P_C (in addition to the fixed effects include in #1 above). Of these 67, 58 came from mammalian populations. As other taxonomic groups were underrepresented and adding another covariate would lead to an over-parametrisation of the

multivariate model, only mammal species were included in this life history analysis.

Finally, as some species were replicated over populations, we fit species as a random effect.

4.3 Results

In total, we analysed vital rate correlations from 136 population matrices comprising 44 species extracted from 39 papers. In circumstances where age-specific fertility or mortality rate remained exactly constant across the entire lifespan, the weighted correlation returned NAs (non-parametric mortality = 10, non-parametric fertility = 15, parametric mortality = 7, non-parametric fertility = 6). For the purposes of visualisation, these NAs replaced with 0s, representing no correlation. However for analysis, these values were omitted as their absolute absence of age-related variances was deemed suspicious.

4.3.1 Assessing the strength of associations between selection and evolutionary predictions

For age-specific mortality, the distribution of Parametric correlations between predicted and observed vital rates revealed three distinct modes: between +0.5 and +1, approximately -0.5, and approximately zero (Fig. 4.1), with a median value of 0.0906. This positive median value suggests that simple evolutionary theories are weakly predictive for mortality rates. For ρ_m, there was a distinct lack of a positive mode, but clear evidence of a trend towards a negative association between observed and predicted fertility values (median ρ_m = -0.246). This negative value suggests a tendency

for evolutionary models to predict a decline in age-specific fertility whilst observed fertility rates in fact increase. Differences between these correlations are quantified by the matrix-specific correlation difference (ρ_{Δ}). The distribution of this measure was positively skewed (skew = 1.396) with a large peak around 0 and a median difference in correlation distributions of 0.0739 confirming that the parametric evolutionary model predicted age-specific mortality slightly better than age-specific fertility.

In comparison, the non-parametric correlations revealed substantially more discrete groupings. ρ_{μ} showed similar, albeit intensified, groupings around +1, -1, and, 0. However, in comparison to the parametric associations, ρ_{m} correlations showed a large negative peak at -1 (Fig. 4.1). The median value for ρ_{μ} was close to zero (median < 0.001), whilst the median ρ_{m} was negative (median = -0.630). Again the overall matrix-specific correlation difference suggested a right skewed (skew = 0.761), more positive distribution, with a median value of 0.117. Suggesting that the non-parametric evolutionary model predicted age-specific mortality slightly better than age-specific fertility. In comparison to the parametric method, which showed an absence of extreme values at -1 and +1, the distribution of the non-parametric correlations suggests a marked contrast in the ability of both methodologies to cope with differences between observed and predicted vital rates. In particular, the non-parametric Spearman's rank ignores the magnitudes of differences in values between the predicted and observed vital rates and ranks them purely on monotonic order. In this way, a population matrix may receive a +1 or -1 purely on the basis of position of rank order, rather than relative difference between values.

Fig. 4.2 reveals the joint distributions of parametric and non-parametric correlations across populations and taxonomic groups. A bootstrapped Kolmogorov-

Smirnov (Sekhon 2011), which measures the degree of similarity (*D*) between two distributions, revealed significant differences between the parametric and non-parametric mortality ($\rho_{\mu}\ D$ = 0.3014, p = <0.001), fertility ($\rho_{m}\ D$ = 0.4176, p = <0.001) and correlation differences analyses ($\rho_{\Delta}\ D$ = 0.2394, p = 0.002). This significant difference between these two methodologies further highlights the statistical distinction between these two approaches. The non-parametric is qualitative, i.e. when selection is high then vital rates should also be high, in comparison, the parametric is quantitative and compares the numerical difference in values between predicted and observed vital rates.

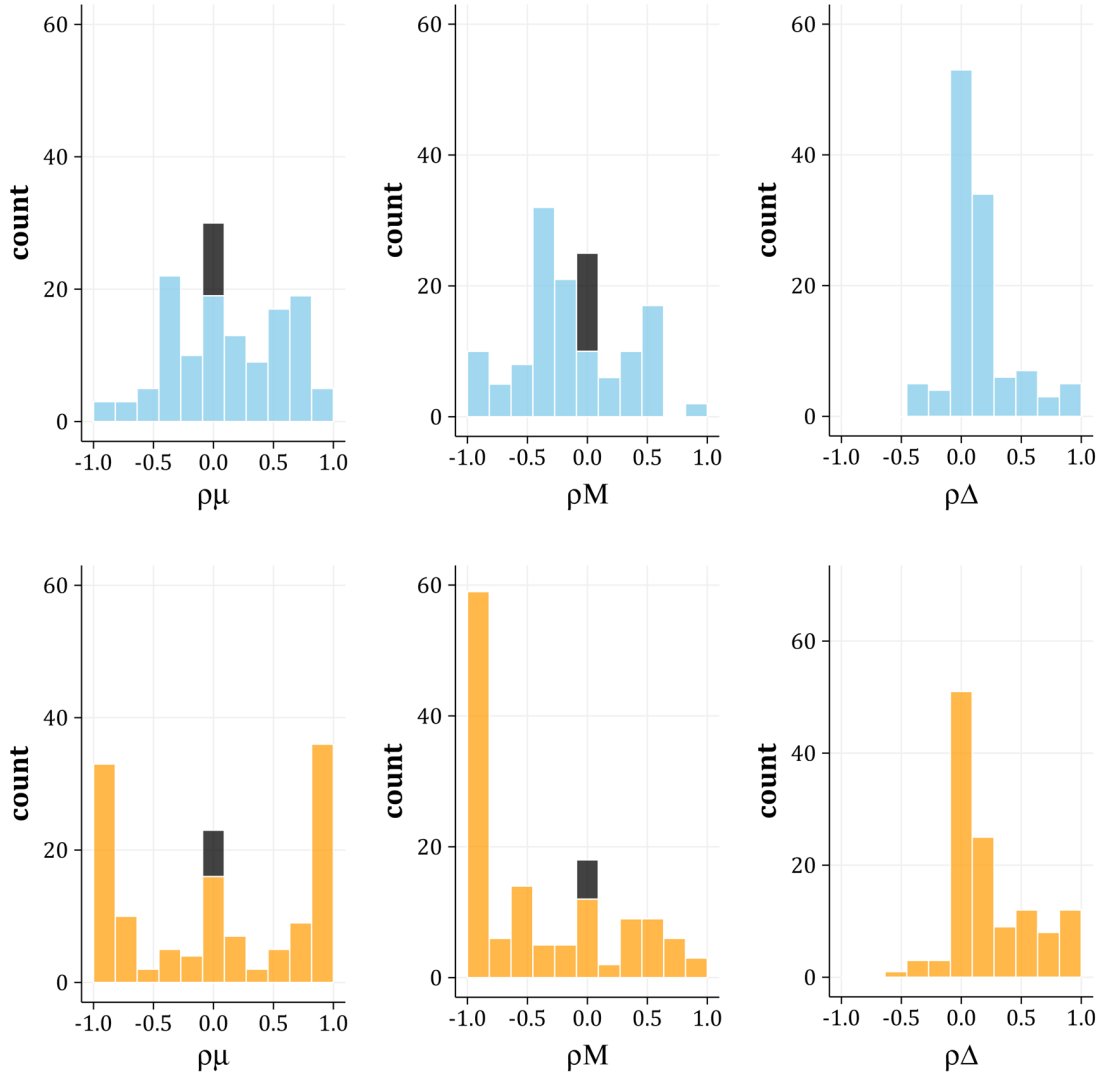

Fig. 4.1 Parametric (top, blue) and non-parametric (bottom, orange) correlations of observed vs predicted vital rates (from Charlesworth's, 1996 and 2001 population genetic models).

Note - The observations in black represent cases where vital rates were reported to be invariant with age.

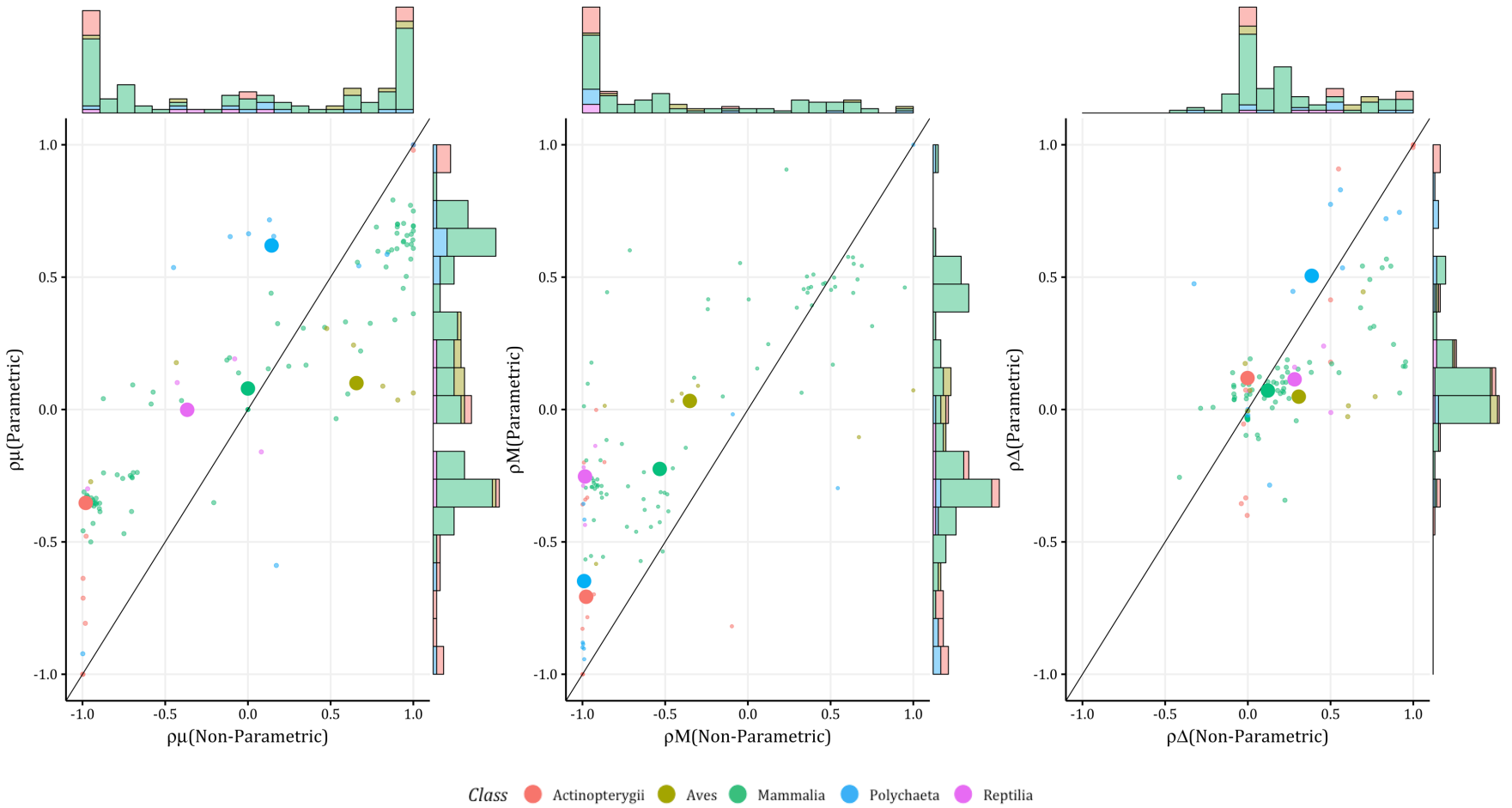

Fig. 4.2 Scatterplots with marginal histograms showing association between the non-parametric and parametric correlations for ρ_{μ}, ρ_{m} and ρ_{Δ}.

Note - The colours on the histogram and points on the scatterplot represent the five predominant taxonomic groups. The larger points represent median correlation values for each taxonomic group. The line represents a simple 1:1 correlation between the two associations.

4.3.2 Impact of life history variables

4.3.2.1 Mortality rates

Using the simple model, we found significantly positive effects of generation time (Parametric β = 0.048, P = 0.002; Non-parametric β = 0.086, P = 0.002) and negative effects of the degree of iteroparity (Parametric β = -0.079, P = 0.024 ; Non-parametric β = -0.173, P = 0.006) upon the performance of the simple evolutionary model to predict patterns of age-specific mortality. This pattern held over both parametric and non-parametric models (Table 4.1). Whilst we might expect heterogeneity to cause a negative effect of increasing degree of iteroparity on fertility correlations, the cause for a relationship between iteroparity on mortality rate correlations is less clear.

Fish and reptiles were associated with poorer fits (Parametric fish β = -0.479, P = 0.002; Non-parametric fish β = -0.605, P = 0.011; Parametric reptiles β = -0.333, P = 0.125; Non-parametric reptiles β = -0.646, P = 0.058) compared to birds and mammals (Parametric birds β = 0.038, P = 0.822; Non-parametric birds β = 0.536, P = 0.041), but these differences were significant only for fish. Whilst the effect sizes for fish and reptiles were similarly negative across both the non-parametric and parametric comparisons, the larger standard errors surrounding the reptile estimate caused the effect to be statistically non-significant. Lastly, whilst not significant, birds showed more positive correlations between observed and predicted vital rates in comparison to other taxa. This suggests that natural selection is acting as a better predictor of avian age-specific fertility and mortality rates in comparison to other taxa, especially fish and reptiles.

Using the life-history model (with mammals only), we found small but insignificant positive effects of generation time on mortality rate correlations estimated by both the

parametric and non-parametric models (Parametric β = 0.011, *P* = 0.582; Non-parametric β = 0.011, *P* = 0.822). This effect was similar, albeit smaller, than the trend observed in the simple model. We observed a similar negative effect of increasing degree of iteroparity on these correlations in parametric and nonparametric models, but this effect was significant only in the parametric analysis (Parametric β = -0.097, *P* = 0.019; Non-parametric β = -0.158, *P* = 0.113) (we note, however, that the effect derived by the non-parametric model was more negative). Both adult body size and parental care duration were positively associated with mortality rate correlations in parametric and nonparametric models. The former effect was significant only in the parametric model (Parametric β = 0.171, *P* = 0.028; Non-parametric β = 0.234, *P* = 0.241), whilst the latter effect was statistically non-significant in both the parametric and non-parametric models (Parametric β = 0.081, *P* = 0.554; Non-parametric β = 0.365, *P* = 0.289) (Table 1).

Table 4.1 Parameter estimates (with standard error), *z* scores and *p* values for the simple and full ρ_μ correlation analysis.

Model	Trait	Parameter	Estimate (SE)	*z* score	*p* value
Simple	Non-parametric ρ_μ	Intercept	-0.100 (0.189)	-0.526	0.599
		T	0.086 (0.028)	3.118	**0.002**
		σ_T	-0.173 (0.063)	-2.726	**0.006**
		Birds	0.536 (0.262)	2.043	**0.041**
		Fish	-0.605 (0.237)	-2.550	**0.011**
		Reptiles	-0.646 (0.341)	-1.892	0.058
		Mammals	-	-	**-**
		Intercept	-0.010 (0.118)	-0.083	0.934

	Parametric ρ_μ	T	0.048 (0.015)	3.135	**0.002**
		σ_T	-0.079 (0.035)	-2.249	**0.024**
		Birds	0.038 (0.169)	0.225	0.822
		Fish	-0.479 (0.153)	-3.125	**0.002**
		Reptiles	-0.333 (0.217)	-1.535	0.125
		Mammals	-	-	**-**
Life-history	Non-parametric ρ_μ	Intercept	-1.099 (0.727)	-1.511	0.131
		T	0.011 (0.047)	0.225	0.822
		σ_T	-0.158 (0.100)	-1.586	0.113
		$\log(M_A)$	0.234 (0.200)	1.173	0.241
		P_C	0.365 (0.344)	1.061	0.289
	Parametric ρ_μ	Intercept	-0.585 (0.285)	-2.054	**0.040**
		T	0.011 (0.020)	0.550	0.582
		σ_T	-0.097 (0.041)	-2.352	**0.019**
		$\log(M_A)$	0.171 (0.078)	2.194	**0.028**
		P_C	0.081 (0.137)	0.592	0.554

4.3.2.2 Fertility rates

The simple model revealed a positive effect of generation time (Parametric β = 0.018, P = 0.230; Non-parametric β = 0.057, P = 0.014) and a negative effect of the degree of iteroparity (Parametric β = -0.063, P = 0.063; Non-parametric β = -0.086, P = 0.105) upon the non-parametric and parametric correlations between predicted and observed fertility rates. We note that whilst the directions of these effects were comparable to

those involving mortality rate correlations, the effect of generation time was statistically significant only in the non-parametric analysis. The smaller effect of increasing degree of iteroparity acting on fertility correlations is consistent with the notion that demographic heterogeneity distorts the evolution of mortality rates away from basic evolutionary predictions more than fertility rates, although why this should be is unclear. In a similar pattern to the mortality rate analysis, fish and reptiles were associated with more negative correlations (Parametric fish β = -0.578, P = <0.001; Non-parametric fish β = -0.534, P = 0.006; Parametric reptiles β = -0.293, P = 0.134; Non-parametric reptiles β = -0.819, P = 0.003), indicating predictions from the evolutionary models fit these animals more poorly than birds and mammals (Parametric birds β = -0.094, P = 0.533; Non-parametric birds β = 0.204, P = 0.341). However, unlike the mortality rate analysis, negative non-parametric effects were significant for both fish and reptiles, although there was a lack of significant effect of reptiles in the parametric correlations.

The life-history model applied to mammals detected a small but insignificant positive effect of generation time on fertility correlations estimated by the non-parametric model (Non-parametric β = 0.037, P = 0.303) and a negative effect estimated by the parametric model (Parametric β = -0.037, P = 0.099). The relative effect sizes were similar to the simple model, however, the increased standard error around these estimates led to non-significance. Again, we observed a similar negative effect of increasing degree of iteroparity on fertility correlations in both models, but these effects were not significant (Parametric β = -0.053, P = 0.257; Non-parametric β = -0.098, P = 0.199). We note, however, that the effect derived by the non-parametric correlations in the life history model was greater than the estimate in the full non-parametric

multivariate analysis. Increasing parental care duration was positively associated with fertility rate correlations in both models, however there was only a significant positive effect in the parametric model (Parametric β = 0.624, *P* = <0.001; Non-parametric β = 0.386, *P* = 0.141). Lastly, adult body size had a small, positive effect on the parametric and non-parametric fertility rate correlations, but these effects were not significant (Parametric β = 0.016, *P* = 0.257; Non-parametric β = 0.000, *P* = 1.000) (Table 4.2). These results, counter to original predictions regarding positive genetic correlations and heterogeneity, suggest that there is an improvement in performance of evolutionary model fit when mammals get heavier and provide parental care for longer durations.

Table 4.2 Parameter estimates (with standard error), *z* scores and *p* values for the simple and full ρ_m correlation analysis

Model	Trait	Parameter	Estimate (SE)	*z* score	*p* value
Simple	Non-parametric ρ_m	Intercept	-0.538 (0.158)	-3.407	**0.001**
		T	0.057 (0.023)	2.460	**0.014**
		σ_T	-0.086 (0.053)	-1.621	0.105
		Birds	0.204 (0.214)	0.952	0.341
		Fish	-0.534 (0.195)	-2.740	**0.006**
		Reptiles	-0.819 (0.279)	-2.936	**0.003**
		Mammals	-	-	**-**
	Parametric ρ_m	Intercept	0.057 (0.105)	0.543	0.587
		T	0.018 (0.015)	1.199	0.230
		σ_T	-0.063 (0.034)	-1.860	0.063
		Birds	-0.094 (0.151)	-0.624	0.533

		Fish	-0.578 (0.136)	-4.266	**<0.001**
		Reptiles	-0.293 (0.195)	-1.499	0.134
		Mammals	-	-	**-**
	Non-parametric ρ_m	Intercept	-0.754 (0.558)	-1.351	0.177
		T	0.037 (0.036)	1.031	0.303
		σ_T	-0.098 (0.077)	-1.284	0.199
		log (M_A)	0.000 (0.152)	0.000	1.000
		P_C	0.386 (0.262)	1.474	0.141
Life-history	Parametric ρ_m	Intercept	-0.272 (0.310)	-0.877	0.380
		T	-0.037 (0.023)	-1.652	0.099
		σ_T	-0.053 (0.047)	-1.133	0.257
		log (M_A)	0.016 (0.087)	0.181	0.856
		P_C	0.624 (0.155)	4.028	**<0.001**

4.3.2.3. Differences between vital rates

The association difference ρ_Δ measures the comparative ability of both evolutionary models to predict patterns of observed age-specific fertility and mortality rates. The simple model revealed a non-significant positive effect of generation time upon the non-parametric and parametric association differences fertility rate correlations (Parametric β = 0.014, *P* = 0.190; Non-parametric β = 0.014, *P* = 0.422). In other words, when generation time increased, there was a weak and statistically insignificant trend for the performance of the simple evolutionary model to predict mortality rates to become increasingly superior to its ability to predict fertility rates. There was also a weak and statistically insignificant negative effect of increasing degree

of iteroparity in the non-parametric model and a positive effect in the parametric correlations (Parametric β = -0.009, P = 0.701; Non-parametric β = 0.028, P = 0.475). Finally, there were no large or significant effects of taxonomic group on association differences (Table 4.3).

When the life history model was considered for mammalian species, there were small, non-significant effects of generation time acting on the non-parametric and parametric association differences, with estimates suggesting both negative and positive trends respectively (Parametric β = 0.021, P = 0.055; Non-parametric β = -0.024, P = 0.429). When the degree of iteroparity increased, both the non-parametric and parametric association difference became increasingly negative (Parametric β = -0.011, P = 0.620; Non-parametric β = -0.004, P = 0.951). This indicates a shift to an improvement in the ability of the evolutionary models to predict patterns of age-specific fertility with increasing degree of iteroparity. These effects were not significantly different from zero, however. Increased adult body size caused both the non-parametric and parametric association differences to shift towards positive values, however this effect was not significant (Parametric β = 0.072, P = 0.051; Non-parametric β = 0.106, P = 0.393). Lastly, whilst increasing duration of parental care had a non-significant, positive effect on the non-parametric association difference, there was a significant negative effect of parental care duration on association differences in the parametric correlations (Parametric β = -0.256, P = <0.001; Non-parametric β = 0.039, P = 0.857). This indicates that when the duration of care increased there was a significant improvement in the ability of the parametric models to predict observed patterns of age-specific fertility rather than morality.

Table 4.3 Parameter estimates (with standard error), *z* scores and *p* values for the simple and full ρ_Δ correlation analysis

Model	Trait	Parameter	Estimate (SE)	*z* score	*p* value
Simple	Non-parametric ρ_Δ	Intercept	0.177 (0.113)	1.571	0.116
		T	0.014 (0.017)	0.803	0.422
		σ_T	-0.028 (0.039)	-0.714	0.475
		Birds	0.183 (0.153)	1.198	0.231
		Fish	-0.004 (0.139)	-0.029	0.976
		Reptiles	0.075 (0.199)	0.378	0.705
		Mammals	-	-	-
	Parametric ρ_Δ	Intercept	-0.071 (0.083)	-0.852	0.394
		T	0.014 (0.011)	1.311	0.190
		σ_T	0.009 (0.024)	0.384	0.701
		Birds	0.078 (0.120)	0.649	0.517
		Fish	0.074 (0.109)	0.681	0.496
		Reptiles	-0.037 (0.154)	-0.244	0.807
		Mammals	-	-	-
Life-history	Non-parametric ρ_Δ	Intercept	-0.163 (0.454)	-0.358	0.720
		T	-0.024 (0.030)	-0.791	0.429
		σ_T	-0.004 (0.063)	-0.061	0.951
		$\log(M_A)$	0.106 (0.124)	0.854	0.393
		P_C	0.039 (0.215)	0.180	0.857
		Intercept	-0.149 (0.132)	-1.123	0.261
		T	0.021 (0.011)	1.918	0.055

Parametric ρ_{Δ}	σ_T	-0.011 (0.023)	-0.495	0.620
	$\log(M_A)$	0.072 (0.037)	1.949	0.051
	P_C	-0.256 (0.069)	-3.709	**<0.001**

4.4 Discussion

We find strong evidence for departures from ageing patterns predicted by simple evolutionary theory amongst the surveyed 44 animal species, and these deviations occurred more frequently for fertility rates than mortality rates. This trend was mirrored in both the non-parametric and parametric analyses, suggesting that these results are robust to violations of the population genetic scaling assumptions made by Charlesworth and Hughes (1996) and Charlesworth (2001). The overall negative relationship between observed and predicted values of age-specific fertility implies that whilst the force of natural selection acting on fertility declines with age, observed age-specific fertility often increases. A more ambiguous trend was observed in age-specific mortality with both deviations and adherences to evolutionary theory, suggesting that overall, natural selection is a very weak predictor of observed mortality patterns. These results, taken together suggest that 1) the underlying genetic architecture for these traits is perhaps more complex than originally assumed by Hamilton, particularly in regards to the extensive deviations observed in age-specific fertility; and 2) other life history constraints, such as selective disappearance, could be contributing to violations from evolutionary theory. Realistically, these two suggestions could both be valid and, whilst we explore potential life history processes below, could suggest that the two population genetic models used in this analysis are underestimating the complexity of trait genetic architecture.

We had expected that the degree of iteroparity would correspond to the opportunity for selective disappearance to influence fertility rates, thereby causing it to be associated with poorer performance of the basic evolutionary models to predict patterns of age-specific fertility. While none of our models found significant effects of iteroparity on these fertility rate correlations, the estimated effect sizes were large and negative and were consistent with this prediction. The lack of statistical significance could be a result of large standard errors surrounding the estimates, suggesting low statistical power and the need for more populations to be added to the analysis.

Intuitively, if the same high-quality females in the population that were biasing fertility correlations also reduced the population's mean mortality risk then we may also expect iteroparity to decrease the value of the basic evolutionary model to predict actuarial senescence. Consistent with this scenario, we found evidence that demographic heterogeneity was significantly affecting the ability of natural selection to predict patterns of age-specific mortality but not fertility, although the direction of the effect was similarly negative for both traits. Whilst the mechanism through which selective disappearance acts on age-specific fertility is clear, with selective mortality of individuals throughout the reproductive period intrinsically linked with individual quality, the larger detected effect size surrounding age-specific mortality requires some clarification. Generation time is the average age of reproduction measured at the population level, and the standard deviation around T quantifies individual variation about that mean (measures are taken from the perspective of offspring). Thereby if the population has a large degree of within-individual variation accompanied with high reproductive costs (resulting in immediate mortality), then we could expect selective

disappearance to have a greater effect on age-specific mortality rather than fertility. Currently, with vital rate data in the form of population-level projection matrices or life tables. we are unable to test this assumption. However, this does highlight the vital importance of longitudinal studies in providing long-term individual-based data for exploring patterns of trait senescence.

Past comparative research has highlighted the large variation that exists in trait ageing trajectories between taxonomic classes, predominantly existing between mammalian and bird species (Williams 1957; Holmes & Austad 1995; Ivimey-Cook & Moorad 2018b). We asked if the basic evolutionary theory of ageing worked in some taxa better than in others. We did find an effect of taxonomic grouping, with the evolutionary models performing significantly worse in reptile and fish species. This trend is to be expected for mortality rates if we consider that reptiles and fish exhibit indeterminate growth, and that can reduce the risk of some sorts of mortality, such as predation risk. This result harmonises with hypotheses from Vaupel *et al.* (2004) who remarked that the occurrence of negligible or negative senescence should be most common in species that attain a non-maximal size at reproductive maturity (Charnov *et al.* 2001; Vaupel *et al.* 2004; Jones *et al.* 2014). As such, we should expect that those taxa that possess this form of development to exhibit ageing trajectories distinct from determinate growers and that do not adhere to predictions from evolutionary theory (Jones *et al.* 2014). Additionally, whilst not significant, birds showed an increased propensity for positive correlations between observed and predicted vital rates in comparison to mammals and other taxa. This suggests that natural selection is acting as a better predictor of avian age-specific fertility and mortality patterns in comparison to other taxa. The underlying reason for

this requires further investigation and a closer, more extensive analysis of avian selection gradients acting on age-specific vital rates.

The contribution of other life history covariates requires explanation too. Firstly, whilst there was no obvious *a priori* reason to expect that increasing generation time would influence correlations, it was a necessary component for the statistical analysis. Held constant, it allowed for inferences to be made regarding variation in degree of iteroparity and comparisons involving other life history traits between animal species where generation times may differ markedly. Increasing generation time had a significant positive effect on both mortality and fertility correlations in the full multivariate model, but this effect disappeared when the model was reduced to only include mammal species. Intuitively this would suggest that, in the full model, the significant effect of the generation time covariate was linked with underlying life history traits typically associated with increasing mean age of reproduction, i.e. adult body mass. Previous comparative research has shown that long generation time was allometrically correlated with large body mass (Gaillard *et al.* 2005, 2008). Similarly, our measure of generation time was positively correlated with adult body mass (+0.857). Therefore, as the full multivariate model contained species from multiple taxa with a wide range of body sizes, from small to very large, the influence of the generation time correlate was amplified and resulted in larger, more statistically significant effect sizes. To clarify the meaning behind the lack of significance in generation time between models, we removed the life history traits (adult body size and parental care duration) and reanalysed the mammalian correlations with only the *T* and degree of iteroparity covariates. The effect of generation time was reduced in the mortality correlations and

only remained significant in the parametric model. For fertility, the effect of generation time increased and remained significant in the non-parametric model. This provides meaningful evidence to suggest that the difference in effect size of generation time, between the simple and life history models, was caused by the positive correlation between *T* and adult body size, and not the removal of other taxonomic classes.

We had expected that increased mammalian body size would have had a negative effect on both fertility and mortality correlations, as bigger organisms would experience increased fertility and lowered mortality with age as susceptibility to predation decreases and capacity to produce more offspring increases (Williams 1966a; Blueweiss *et al.* 1978; Lindstedt & Boyce 1985). Instead, we found that increasing body size had a positive effect on both the mortality and fertility correlations, though only the mortality estimates were statistically significant. This suggests that as mammalian body size increased, the predictions from evolutionary theory were more likely to match observed vital rate trajectories, i.e. decreasing fertility and increasing mortality. The explanation for this trend is unclear, however we can postulate that as increased body mass positively correlates with gestation time (Blueweiss *et al.* 1978), then perhaps declining fertility with increasing adult size is a result of high reproductive costs and an inability to continuously reproduce into older ages, unlike species with indeterminate growth. Additionally, whilst previous theory led us to expect that increasing body size would lead to a decrease in mortality risk and progressively more negative correlations, the shift towards more positive correlations suggests that this particular phenomenon was not occurring. Instead, the evolutionary models predicting age-specific mortality better with increased body size. This positive trend is to be expected if organisms suffer

increased rate of mortality as a result of the faster growth rate needed to attain a larger body size through the need to search for more resources and undertake riskier foraging strategies, or experience reduced survival whilst at a larger body size due to an increase in predation resulting from increased visibility and lower agility (See Table 1, Blanckenhorn 2000). Future research should focus on comparatively reviewing rates of age-specific mortality and fertility in species with varying growth rates, to see if these assumptions regarding increased mortality and reduced fertility are met.

Lastly, we had expected that if positive genetic correlations existed between extended late-age lifespan and social care, then species with prolonged periods of parental care would exhibit poorer fits to evolutionary predictions. We found no evidence to support this hypothesis; instead, we found evidence to suggest that increased parental care caused observed vital rates to fit better with predicted values from evolutionary models. However, this effect was only significant when observing parametric fertility correlations. This is consistent with cross-generational negative genetic correlations across ages existing between extended social care and traits vital to fitness, such as those expected from the reproductive effort hypothesis (Charlesworth & Leon 1976; Nussey *et al.* 2008b; Boonekamp *et al.* 2014). As such, this result is consistent with fitness costs associated with trade-offs between enhanced social care of offspring and parental survival and fertility at late ages. In order to formally test for the presence of such a cross-generational trade-off, it would be necessary to perform a quantitative genetics analysis on longitudinal, individual-based data in order to assess the phenotypic and genetic correlation between early offspring survival and parental survival/fertility. A similar use of quantitative genetics to investigate the pleotropic

effects of age of first and last reproduction was conducted in mute swans (*Cygnus olor*) (See Charmantier *et al.* 2006). In particular, they found compelling evidence for a genetically-linked trade-off between increased early-life performance and faster late-life decline in reproduction (Charmantier *et al.* 2006)

Overall, this result provides tentative support for both the antagonistic pleiotropy (Williams 1957) and disposable soma (Kirkwood 1977) theories of ageing, whilst different in their mechanisms, suggest that increased investment in reproduction in early-life leads to an increased rate of senescence in late-life. This result adds to the growing collection of evidence to suggest that this early/late-life trade-off is a common occurrence in mammalian species, where high reproductive investment in early life is accompanied with faster rates of reproductive and actuarial senescence in late-life (Nussey *et al.* 2006; Lemaître *et al.* 2015)

4.5 Conclusion

We provide the first extensive and comparative review of violations from Hamilton-like ageing across multiple animal species. In particular, we provide compelling evidence to suggest that biological constraints are readily contributing to the distortion of observed ageing patterns and leading to departures from evolutionary predictions. Furthermore, we find convincing support to suggest that fertility senescence appears to be more sensitive to these constraints than actuarial senescence and that processes such as demographic heterogeneity and indeterminate growth are significantly affecting the predictive performance of evolutionary theory. Taken together, we highlight the critical importance in needing to further understand the nature of these life history phenomena

and other processes that readily interfere with the ability of natural selection to predict vital rate ageing trajectories. In doing so, we will vastly increase our knowledge about the diversity of ageing patterns that exist across the tree of life.

Chapter 5: General Discussion

5.1 Thesis Overview

This thesis addressed the following outstanding questions:

5.1.1 How are maternal age effects distributed across multiple taxa?

Despite extensive comparative work describing the vast observed diversity that exists for actuarial and reproductive ageing trajectories (Promislow & Harvey 1991; Ricklefs 1998; Ricklefs & Scheuerlein 2001; Ricklefs *et al.* 2003; Jones *et al.* 2008, 2014; Péron *et al.* 2010; Lemaître *et al.* 2013; Nussey *et al.* 2013; Lemaître & Gaillard 2017), the distribution of maternal age effects across the tree of life remains unclear (Bloch Qazi *et al.* 2017). For this reason, I employed both experimental and meta-analytical techniques to thoroughly review the occurrence and intensity of maternal effect senescence across multiple taxa. In particular I identified whether maternal age was an important predictor of offspring fitness in both laboratory and natural populations. Lastly, I provided the most appropriate experimental design to date in order to study the individual longitudinal effects of pre- and postnatal maternal age in the burying beetle, *Nicrophorus vespilloides*.

5.1.2 How does a knowledge of natural selection and evolution help us to understand the observed diversity in maternal age effects and demographic senescence?

Whilst we can appreciate there is great diversity in trait ageing trajectories, described in detail by previous comparative work (see Jones *et al.* 2014), the ultimate evolutionary cause for this variation remains vague. To this end, I extensively tested multiple

evolutionary and population genetic theories (Hamilton 1966; Charlesworth & Hughes 1996; Charlesworth 2001; Moorad & Nussey 2016) in their ability to predict patterns of trait senescence. In particular, I asked, to what extent was natural selection contributing to the evolution of trait senescence. I also identified potential environmental and life history processes that were contributing to non-conformance to evolutionary theory.

5.2 Key Findings

5.2.1 Chapter 2: Experimental insights

In Chapter 2, I found that neither the deleterious effects of increasing maternal age (separated into the individual components of egg-producer and carer age) nor reproductive effort increases appeared to manifest when measuring traits at the level of the offspring or female. This is counter to evolutionary predictions made by Moorad and Nussey (2016) regarding maternal effect senescence and those made by reproductive effort models (Williams 1966a; Hirshfield & Tinkle 1975; Charlesworth & Leon 1976; Clutton-Brock 1984). Furthermore, I found no evidence to suggest that the selective disappearance of carers was contributing to bias in our results. These results suggest that either 1) predictions from evolutionary and life history theory do not always manifest, or 2) the decrease associated with senescence and the increase associated with reproductive effort are exactly equal and cancel each other out. This Chapter highlights that perhaps current theory is insufficient to account for the true diversity in ageing patterns that we observe.

5.2.2 Chapter 3: Maternal effect senescence

In Chapter 3, I used meta-analytical techniques to extensively review and analyse the distribution of maternal age effects acting on neonatal survival in 90 replicates of 51 laboratory, semi-captive and wild animal species. Firstly, I compared the performance of age-independent, linear and quadratic models of maternal age. I firstly provided overwhelming evidence to suggest that maternal age was an important predictor of neonatal survival (important in 91% of cases). In particular, I offered convincing evidence that the quadratic form of maternal age was the best predictor of neonatal survival (preferred in 65 out of 90 replicates). Crucially, I found that the linear models of maternal age tended to underestimate the true signature of maternal effect senescence as often, maternal effect senescence accelerated with age (as predicted by Moorad and Nussey 2016). As a result, I then reassessed the effect of maternal age in post-generation time, old-age females. I found strong evidence of maternal effect senescence in laboratory and wild mammal species (-67.1% and -57.8%, per standardized unit of increasing age) but a much reduced effect in wild bird species (-0.7%, per standardized unit of increasing age). Suggesting significant taxonomic differences in maternal effect ageing rates. Next, I asked how well did evolutionary theory (from Moorad and Nussey, 2016) explain the patterns of maternal effect senescence that we observed. In particular, I found that the evolutionary model showed vast predictive improvements when describing patterns of maternal effect senescence in natural, wild populations in comparison to laboratory populations. This result, taken together, suggests that natural selection is a contributing factor to the shape and diversity of maternal effect ageing patterns in wild populations.

5.2.3 Chapter 4: Vital rates and evolutionary theory

In Chapter 4, I wanted to further investigate whether natural selection was a causal determinant of the diversity in ageing patterns relating to senescence. As previous research has focused extensively on describing the observed diversity in ageing patterns, particularly age-specific survival and fertility (see Jones *et al.* 2014), this chapter sought to better understand the ultimate underlying cause for this variation by comparatively assessing the distribution of adherences and violations to predictions from evolutionary theory provided by William Hamilton (1966) across multiple animal species. Additionally, like with Chapters 2 and 3, I wanted to identify life history processes, such as selective disappearance, that were contributing to non-conformance to evolutionary theory. In fact, I found widespread evidence of departures from evolutionary theory, particularly when observing age-specific fertility. This implies that often, when the force of natural selection acting on fertility declines with increasing age (Hamilton 1966), observed age-specific fertility was actually increasing. This results suggest that perhaps age-specific fertility is more complex than originally assumed by Hamilton, and that fertility senescence is affected to a greater degree by biological phenomena in comparison to actuarial senescence. Lastly, I identified significant taxonomic differences in prevalence of departures from predicted evolutionary endpoints in reptiles and fish (species that show indeterminate growth). Additionally, I found tentative evidence to suggest that demographic heterogeneity or selective disappearance was influencing population adherence to evolutionary predictions. Taken together, this chapter provides significant evidence to suggest that the predictions of natural selection, particularly pertaining to

fertility senescence and reptile/fish species, are readily distorted by interfering biological processes.

5.3 Implications and Future Directions

5.3.1 The force of natural selection acting on senescing traits

The results presented in Chapters 3 and 4 provide convincing evidence that natural selection and evolutionary forces are contributing to the vast diversity of observed ageing rates. In particular, these Chapters show that traits relating to senescence, namely, survival, reproduction and more recently theorised, maternal effect senescence (Moorad & Nussey 2016), are likely to have been shaped by natural selection. Whilst this work adds to the growing number of studies describing the age-related trajectories of senescent traits, these results also provide compelling insight to suggest that the ultimate underlying cause for this variation, particularly apparent in wild populations is produced from biological processes distorting the force of natural selection. We note that as the evolution of trait senescence is inherently reliant on the underlying genetic architecture for which natural selection can act upon, there is a need for more in depth, quantitative genetic analyses, particularly applied to different taxonomic groups from wild populations. Specifically as evolutionary theories often make assumptions regarding genetic architecture that may not always be met, such as the lack of age-specific genetic variation for maternal effects in Chapter 2 or the reduced maternal effect signature found in birds in Chapter 3, or that are perhaps too simplistic, such as the large violations from evolutionary theory in Chapter 4.

5.3.2 Life history processes

Throughout this thesis I have identified several life history phenomena and biological processes that readily distort ageing trajectories away from the decline predicted by natural selection and evolutionary theory (Hamilton 1966). In particular, Chapter 4 provided evidence to suggest that demographic heterogeneity and an organism's life history (such as indeterminate vs determinant growers) were contributing to non-conformance to predicted declines. This highlights the need for further study investigating species with unique life histories, present in such taxa as reptiles and fish. Additionally in this Chapter, I highlighted processes that could be contributing to increased conformance and exacerbating senescent declines, such as the presence of cross-generational negative correlations, consistent with early life increases in reproductive effort resulting in late-life trade-offs in fertility or survival. In Chapter 3, I highlight the potential problems faced by comparative assessments of senescence when these biological processes are not accounted for. Specifically, I assert that selective disappearance might be one of the predominant underlying causes for a reduced maternal effect senescence signature experienced in birds. This result is in stark contrast to the trend observed in Chapter 4 where predicted evolutionary declines for age-specific mortality and fertility were more closely matched in birds than other taxa. Whilst these results warrant further investigation, we can speculate that this suggests a possible link between selective disappearance and quality of maternal care. Therefore, I suggest that future studies, especially those investigating maternal effect ageing or the ageing trajectories of traits, to simply incorporate the fitting of time-of-death into

standard statistical models in order to get a true, unbiased longitudinal view of trait senescence. As such, in Chapter 2, I offer an experimental design that allows for the clearest estimate of maternal age effects to be measured in laboratory populations, by statistically controlling for demographic heterogeneity and a thoroughly sampling "old" maternal ages whilst controlling for the confounding effects of multiple reproductive attempts.

5.4 Concluding remarks

The work presented here represents a comprehensive comparative assessment of the contribution of natural selection and several other biological processes in shaping trait ageing trajectories across multiple animal species. Chapters 3 and 4 provide convincing evidence to suggest that natural selection is a contributing factor in shaping ageing trajectories in wild populations and that biological processes such as demographic heterogeneity lead to an underestimation of trait senescence and departures from evolutionary theory. To this end, Chapter 2 provides the most appropriate experimental design to date to allow for the individual components of maternal effect ageing to be measured whilst accounting for the bias of selective disappearance of carers.

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Appendices

Supplementary material from: "Disentangling pre- and postnatal maternal age effects on offspring performance in an insect with elaborate maternal care"

Table S2.1 Likelihood ratio tests for multivariate carer-level models with and without block effects

Trait	Log-likelihood with block	Log-likelihood without block	*D*	*p* value
Larval weight at dispersal	196.560	195.871	1.378	0.240
Residual lifespan of carer	-183.301	-183.726	0.850	0.357
Weight change of carer	125.030	125.030	0.000	1.000
Number of larvae surviving to dispersal	-88.8348	-91.169	4.668	**0.031**

Table S2.2 Summary of models (null, linear and quadratic) for each trait at the level of the offspring and the carer used in model selection

Trait	Model	Log-likelihood	*k*	AIC
Larval weight at dispersal	Fixed: Carcass weight + age of carer at death Random: Block/carer ID	1330.058	8	-2644.117
	Fixed: Carer age + egg-producer age + carcass weight + age of carer at death Random: Block/carer ID	1331.342	10	-2642.683
	Fixed: Carer age^2 + egg-producer age^2 + carer age*egg-producer age + carcass weight + age of carer at death **Random: Block/carer ID**	**1336.983**	**13**	**-2647.966**
Offspring adult longevity	**Fixed: Carcass weight + age of carer at death** **Random: Block/carer ID**	**-2447.873**	**8**	**4911.746**

	Fixed: Carer age + egg-producer age + carcass weight + age of carer at death Random: Block/carer ID	-2447.480	10	4914.96
	Fixed: Carer age^2 + egg-producer age^2 + carer age*egg-producer age+ carcass weight + age of carer at death Random: Block/carer ID	-2444.582	13	4915.163
Larval weight at hatching	**Fixed: Carcass weight + age of carer at death**	**191.207**	**5**	**-372.415**
	Fixed: Carer age + egg-producer age + carcass weight + age of carer at death	192.284	7	-370.567
	Fixed: Carer age^2 + egg-producer age^2 + carer age*egg-producer age+ carcass weight + age of carer at death	192.994	10	-365.987
	Fixed: Carcass weight + age of carer at death	-251.987	5	513.974

Residual lifespan of carer	**Fixed: Carer age + egg-producer age + carcass weight + age of carer at death**	**-235.773**	**7**	**485.546**
	Fixed: Carer age^2 + egg-producer age^2 + carer age*egg-producer age + carcass weight + age of carer at death	-233.213	10	486.427
Weight change of carer	**Fixed: Carcass weight + age of carer at death**	**110.630**	**5**	**-211.259**
	Fixed: Carer age + egg-producer age + carcass weight + age of carer at death	112.399	7	-210.798
	Fixed: Carer age^2 + egg-producer age^2 + carer age*egg-producer age+ carcass weight + age of carer at death	112.738	10	-205.476
Number of larvae surviving to dispersal	Fixed: Carcass weight + age of carer at death Random: Block	-130.747	6	273.494
	Fixed: Carer age + egg-producer age + carcass weight + age of carer at death	-129.496	8	274.992

Random: Block			
Fixed: Carer age^2 + egg-producer age^2 + carer age*egg-producer age + carcass weight + age of carer at death	**-123.552**	**11**	**269.105**
Random: Block			

Note - The best model for each trait is shown in bold-face.

Table S2.3(a) Parameter estimates from the full multivariate linear model assessed at the level of the carer**.**

Trait	Variables	*Effect Size Estimate*	Standard Error	*z-score*	*p* value
Larval weight at hatching (mg)	**Intercept**	**49.400**	**19.700**	**2.508**	**0.012**
	Carer age	-0.077	0.056	-1.363	0.173
	Egg-producer age	-0.035	0.051	-0.688	0.491
	Carcass weight	-0.240	0.788	-0.305	0.761
	Age of carer at death (2 to 5)	-4.710	7.420	-0.635	0.526
	Age of carer at death (8 to 11)	4.970	2.830	1.756	0.079
	Age of carer at death (11+)	0.000	0.000	-	-
Residual lifespan of carer (days)	Intercept	114.600	63.400	1.808	0.071
	Carer age	**-1.122**	**0.182**	**-6.165**	**<0.001**
	Egg-producer age	-0.107	0.163	-0.656	0.512
	Carcass weight	0.291	2.537	0.115	0.909
	Age of carer at death (2 to 5)	**-85.580**	**23.860**	**-3.587**	**<0.001**

	Age of carer at death (8 to 11)	**-50.870**	**9.103**	**-5.588**	**<0.001**
	Age of carer at death (11+)	0.000	0.000	-	-
Weight change of carer (mg)	Intercept	40.200	88.900	0.452	0.651
	Carer age	0.394	0.255	1.545	0.122
	Egg-producer age	-0.097	0.229	-0.425	0.671
	Carcass weight	-1.330	3.560	-0.374	0.709
	Age of carer at death (2 to 5)	29.700	33.500	0.887	0.375
	Age of carer at death (8 to 11)	16.800	12.800	1.313	0.189
	Age of carer at death (11+)	0.000	0.000	-	-
Number of larvae surviving to dispersal	Intercept	7.955	8.136	0.978	0.328
	Carer age	-0.032	0.025	-1.258	0.208
	Egg-producer age	-0.028	0.023	-1.206	0.228
	Carcass weight	0.217	0.326	0.666	0.506
	Age of carer at death (2 to 5)	-2.697	2.923	-0.923	0.356
	Age of carer at death (8 to 11)	-0.028	1.396	-0.020	0.984

Age of carer at death (11+)	0.000	0.000	-	-
Block 1	1.035	1.175	0.881	0.378
Block 2	0.493	1.114	0.443	0.658
Block 3	0.911	1.096	0.831	0.406
Block 4	0.512	1.284	0.399	0.690
Block 5	-1.216	1.192	-1.02	0.308
Block 6	0.255	1.141	0.223	0.823
Block 7	1.490	1.681	0.886	0.375
Block 8	0.627	1.653	0.379	0.704
Block 9	**-4.108**	**1.253**	**-3.279**	**0.001**

Table S2.3(b) Parameter estimates from the full multivariate quadratic model assessed at the level of the carer.

Trait	Variables	*Effect Size Estimate*	Standard Error	*z-score*	*p* value
Larval weight at hatching (mg)	**Intercept**	**42.6700**	**21.6100**	**1.975**	**0.048**

	Carer age^2	-0.0003	0.0029	-0.108	0.914
	Egg-producer age^2	-0.0025	0.0031	-0.823	0.411
	Carer age* egg-producer age	-0.0033	0.0037	-0.887	0.375
	Carer age	0.0385	0.3201	0.120	0.904
	Egg-producer age	0.2667	0.3224	0.827	0.408
	Carcass weight	-0.2219	0.8128	-0.273	0.785
	Age of carer at death (2 to 5)	-6.5510	7.7660	-0.844	0.399
	Age of carer at death (8 to 11)	4.5180	3.0020	1.505	0.132
	Age of carer at death (11+)	0.0000	0.0000	-	-
Residual lifespan of carer	Intercept	87.950	67.150	1.310	0.190
	Carer age^2	0.005	0.009	0.550	0.582
	Egg-producer age^2	-0.013	0.010	-1.358	0.174
	Carer age* egg-producer age	-0.014	0.011	-1.214	0.225
	Carer age	-1.163	0.995	-1.169	0.242
	Egg-producer age	1.356	1.002	1.353	0.176
	Carcass weight	0.530	2.526	0.210	0.834

	Age of carer at death (2 to 5)	**-97.160**	**24.130**	**-4.027**	**<0.001**
	Age of carer at death (8 to 11)	**-54.540**	**9.328**	**-5.847**	**<0.001**
	Age of carer at death (11+)	0.000	0.000	-	-
Weight change of carer (mg)	Intercept	36.8100	98.2200	0.375	0.708
	Carer age^2	0.0060	0.0131	0.462	0.644
	Egg-producer age^2	-0.0012	0.0139	-0.089	0.929
	Carer age* egg-producer age	-0.0033	0.0168	-0.199	0.842
	Carer age	-0.0309	1.4550	-0.021	0.983
	Egg-producer age	0.1095	1.4660	0.075	0.940
	Carcass weight	-1.0210	3.6950	-0.276	0.782
	Age of carer at death (2 to 5)	24.7500	35.3000	0.701	0.483
	Age of carer at death (8 to 11)	14.0600	13.6500	1.030	0.303
	Age of carer at death (11+)	0.0000	0.0000	-	-
	Intercept	-2.174	8.312	-0.262	0.794
	Carer age^2	-0.002	0.001	-1.509	0.131
	Egg-producer age^2	**-0.003**	**0.001**	**-2.300**	**0.021**

	Carer age* egg-producer age	**-0.004**	**0.001**	**-3.065**	**0.002**
Number of larvae surviving to dispersal	Carer age	0.220	0.120	1.832	0.067
	Egg-producer age	**0.319**	**0.121**	**2.643**	**0.008**
	Carcass weight	0.261	0.303	0.859	0.390
	Age of carer at death (2 to 5)	-4.092	2.765	-1.480	0.139
	Age of carer at death (8 to 11)	-0.600	1.311	-0.457	0.647
	Age of carer at death (11+)	0.000	0.000	-	-
	Block 1	1.48	1.146	1.291	0.197
	Block 2	0.1987	1.089	0.182	0.855
	Block 3	0.803	1.054	0.762	0.446
	Block 4	0.6729	1.243	0.541	0.588
	Block 5	-1.509	1.158	-1.303	0.193
	Block 6	0.8114	1.11	0.731	0.465
	Block 7	1.347	1.631	0.826	0.409
	Block 8	0.1444	1.608	0.090	0.928
	Block 9	**-3.949**	**1.394**	**-2.833**	**0.005**

Table S2.4 Analysis of comparable *Nicrophorus vespilloides* studies

Study	Trait	Effect Size
Ward (2009)	Average larval dispersal weight	-0.00196g/day
		-1.97mg/day
Ward (2009)	Larval survival to dispersal	-0.175 larvae/day
		-0.0087 1/day (brood of 20)
Cotter (2010)	Average total weight of brood	-0.0307g/day
		30.7mg/day

Note - Direct estimates were not provided by Ward (2009). Instead, data were extracted from graphical representations of data. A generalised linear model, weighted by the inverse of the standard error, was performed that regressed the extracted average dispersal weight against the age of mothers (in days). A binomial dataset was constructed using the number of offspring out of an initial brood of 20 that survived or died upon reaching dispersal. The probability of survival was subsequently regressed on age of the mother in a binomial generalised linear model. For brood weight, the same extraction and analysis was repeated for Cotter et al (2010). Average total weight of brood was then regressed against age in days of the mother.

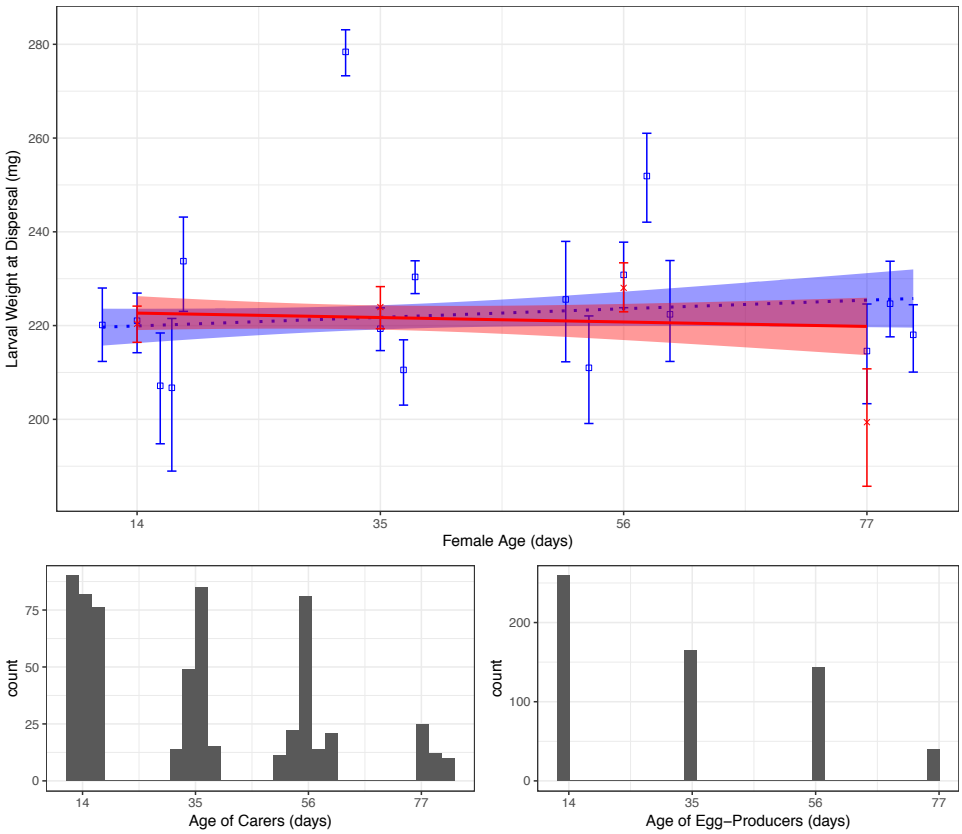

Fig. S2.1(a) Linear fit of larval weight at dispersal to the age of carers (blue dotted line with open squares) and egg-producers (red solid line with crosses).

Histograms below the main plot represents the number of larvae produced by age-specific carers and egg-producers. Smoothed lines signify predicted values from linear or quadratic models. The coloured areas show calculated 95% confidence intervals. Error bars represent standard errors.

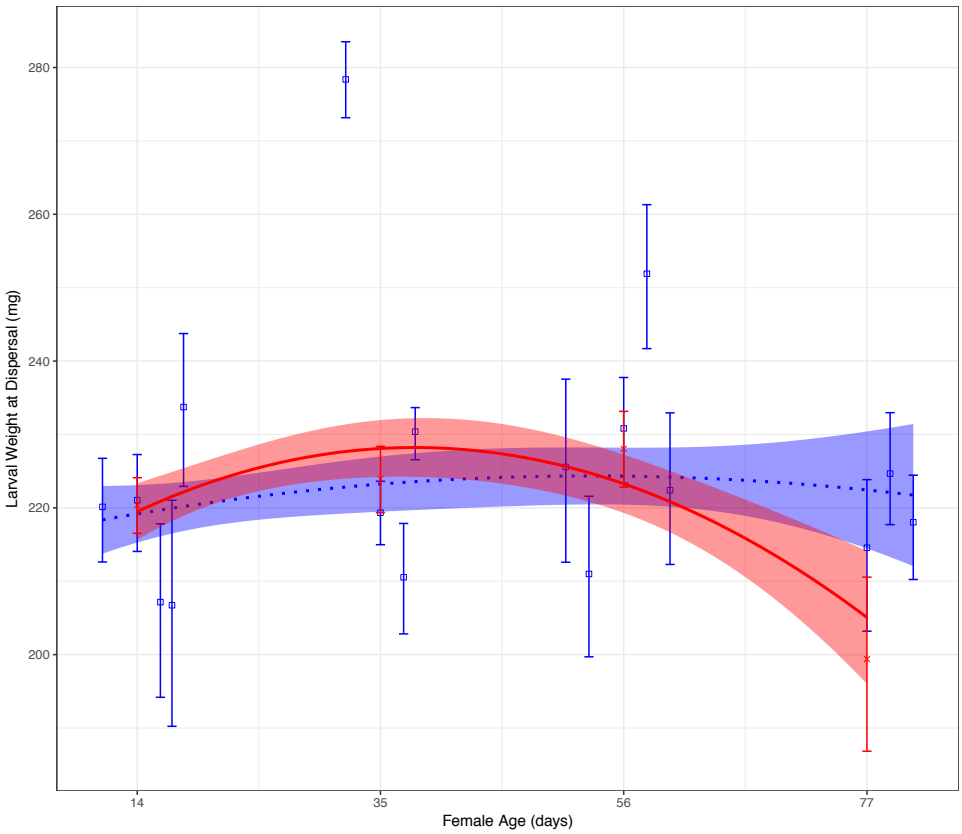

Fig. S2.1(b) Quadratic fit of larval weight at dispersal to the age of carers (blue dotted line with open squares) and egg-producers (red solid line with crosses). Smoothed lines signify predicted values from linear or quadratic models. The coloured areas show calculated 95% confidence intervals. Error bars represent standard errors.

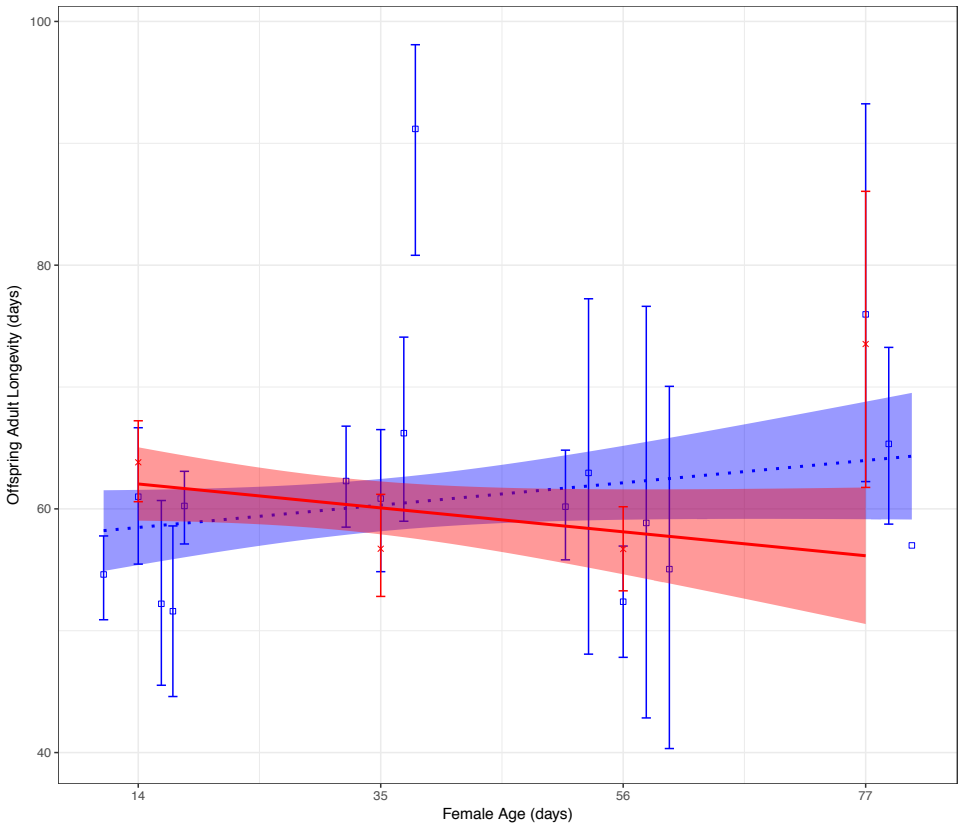

Fig. S2.2(a) Linear fit of offspring adult longevity to the age of carers (blue dotted line with open squares) and egg-producers (red solid line with crosses). Smoothed lines signify predicted values from linear or quadratic models. The coloured areas show calculated 95% confidence intervals. Error bars represent standard errors.

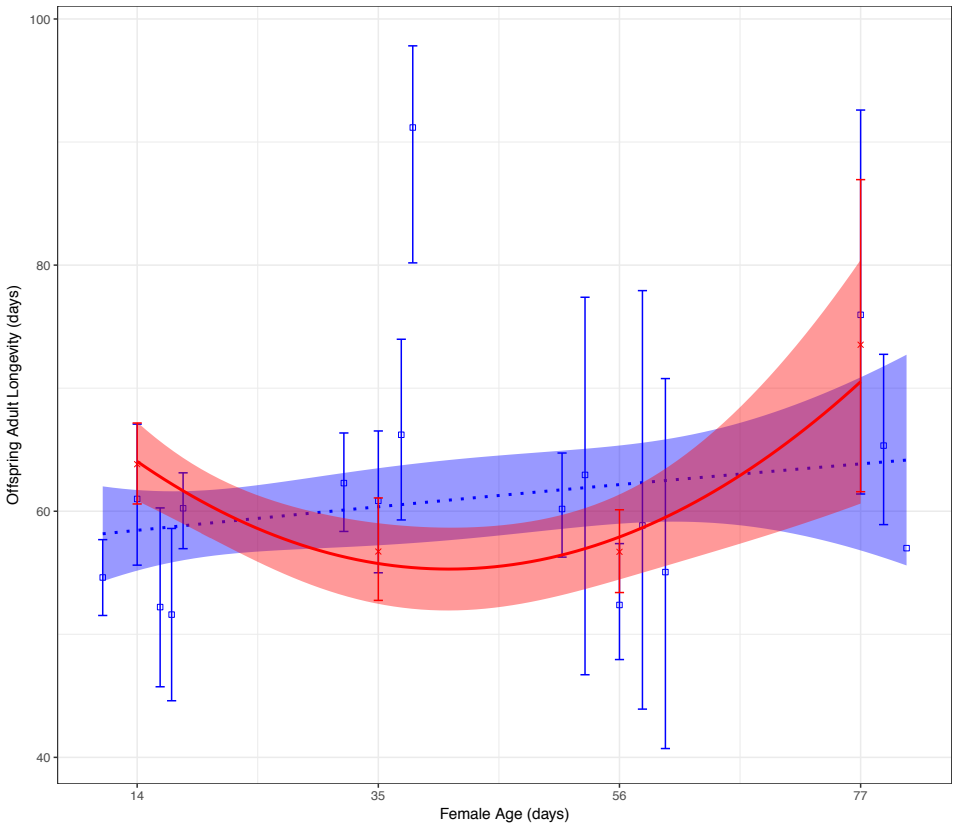

Fig. S2.2(b) Quadratic fit of offspring adult longevity to the age of carers (blue dotted line with open squares) and egg-producers (red solid line with crosses). Smoothed lines signify predicted values from linear or quadratic models. The coloured areas show calculated 95% confidence intervals. Error bars represent standard errors.

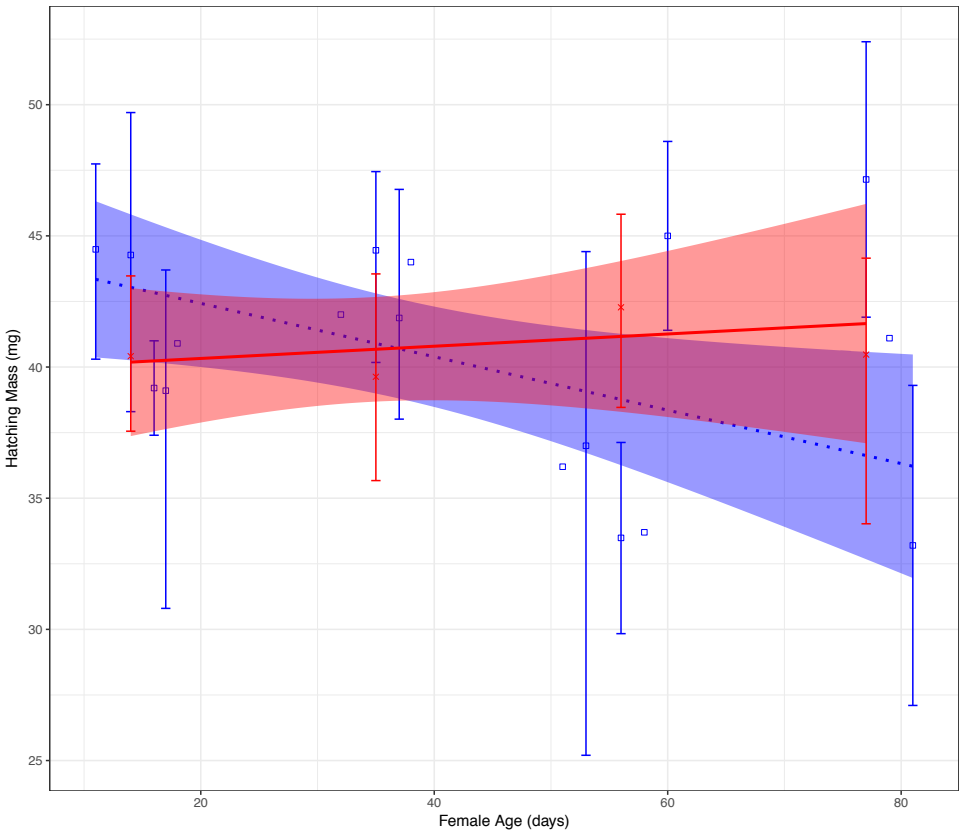

Fig. S2.3(a) Linear fit of larval weight at hatching to the age of carers (blue dotted line with open squares) and egg-producers (red solid line with crosses). Smoothed lines signify predicted values from linear or quadratic models. The coloured areas show calculated 95% confidence intervals. Error bars represent standard errors.

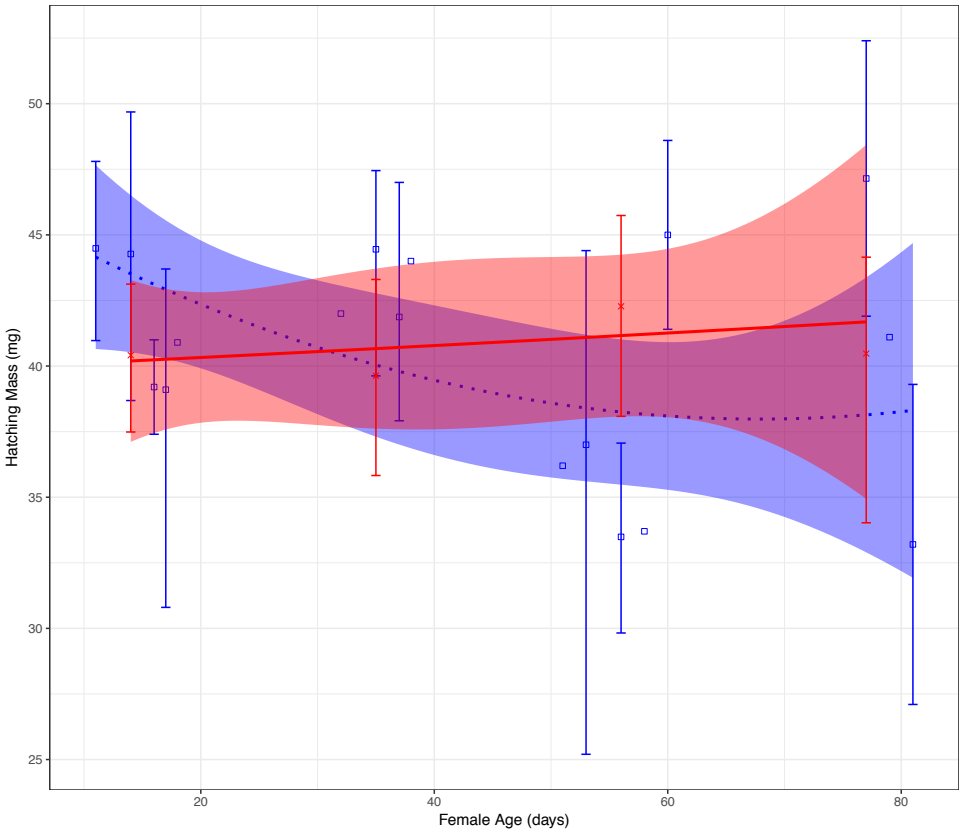

Fig. S2.3(b) Quadratic fit of larval weight at hatching to the age of carers (blue dotted line with open squares) and egg-producers (red solid line with crosses). Smoothed lines signify predicted values from linear or quadratic models. The coloured areas show calculated 95% confidence intervals. Error bars represent standard errors.

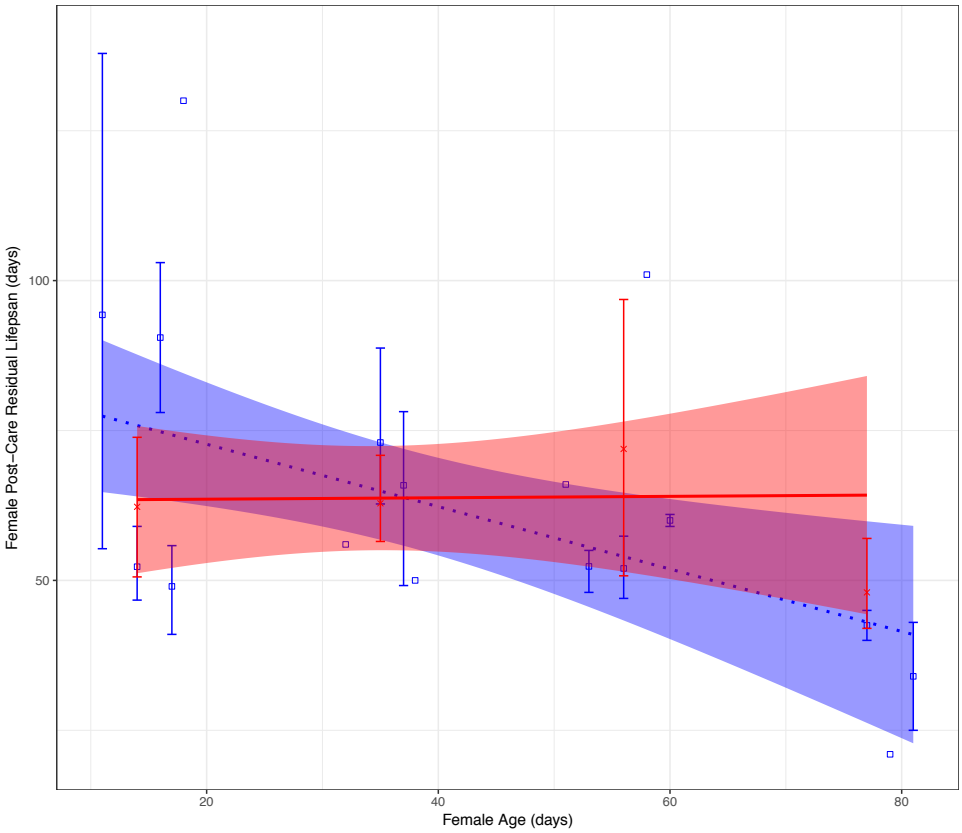

Fig. S2.4(a) Linear fit of female post-care residual lifespan to the age of mating (blue dotted line with open squares) and egg-producers (red solid line with crosses). Smoothed lines signify predicted values from linear or quadratic models. The coloured areas show calculated 95% confidence intervals. Error bars represent standard errors.

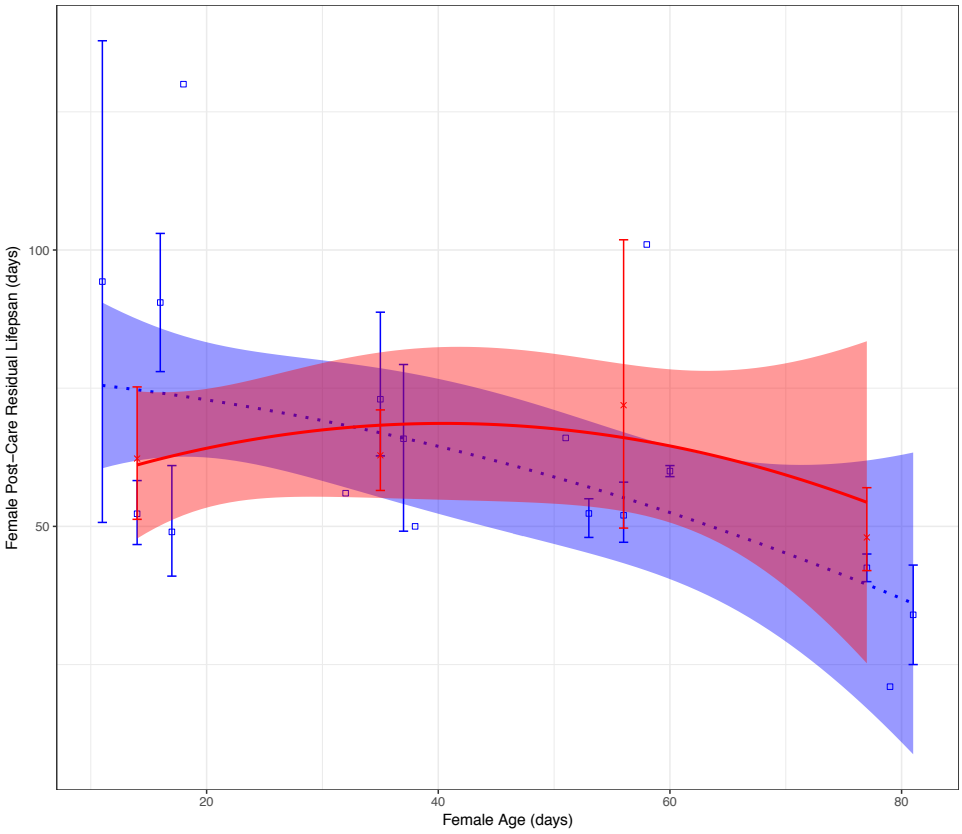

Fig. S2.4(b) Quadratic fit of female post-care residual lifespan to the age of mating (blue dotted line with open squares) and egg-producers (red solid line with crosses). Smoothed lines signify predicted values from linear or quadratic models. The coloured areas show calculated 95% confidence intervals. Error bars represent standard errors.

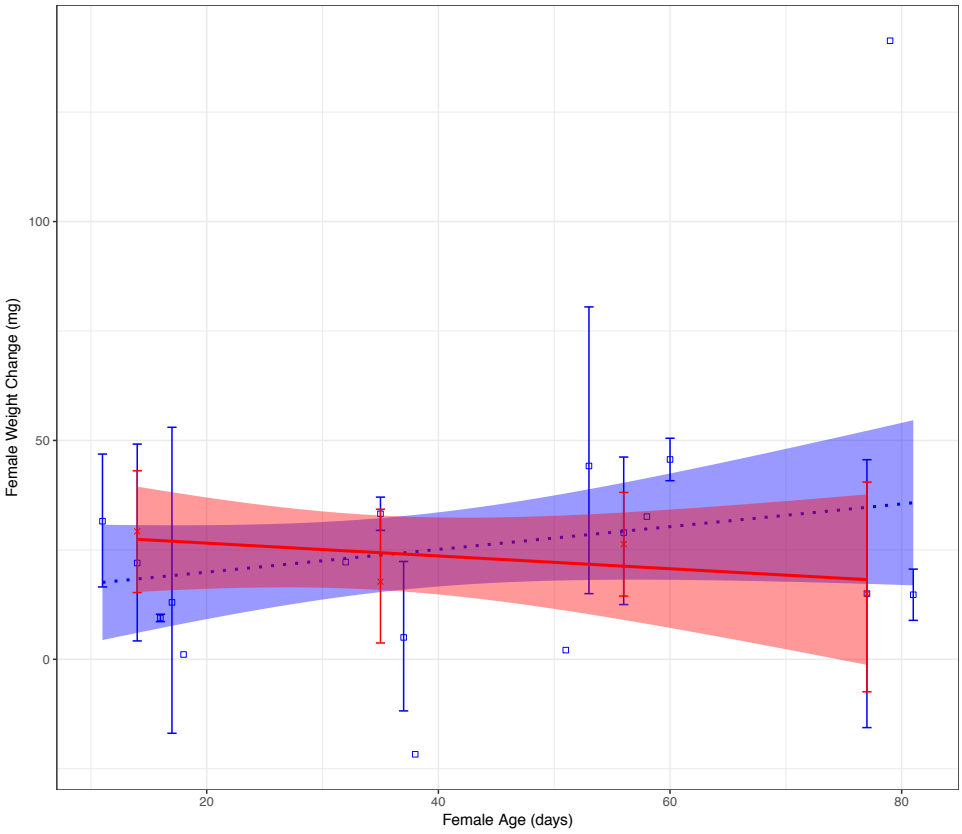

Fig. S2.5(a) Linear fit of carer weight change to the age of mating (blue dotted line with open squares) and egg-producers (red solid line with crosses). Smoothed lines signify predicted values from linear or quadratic models. The coloured areas show calculated 95% confidence intervals. Error bars represent standard errors.

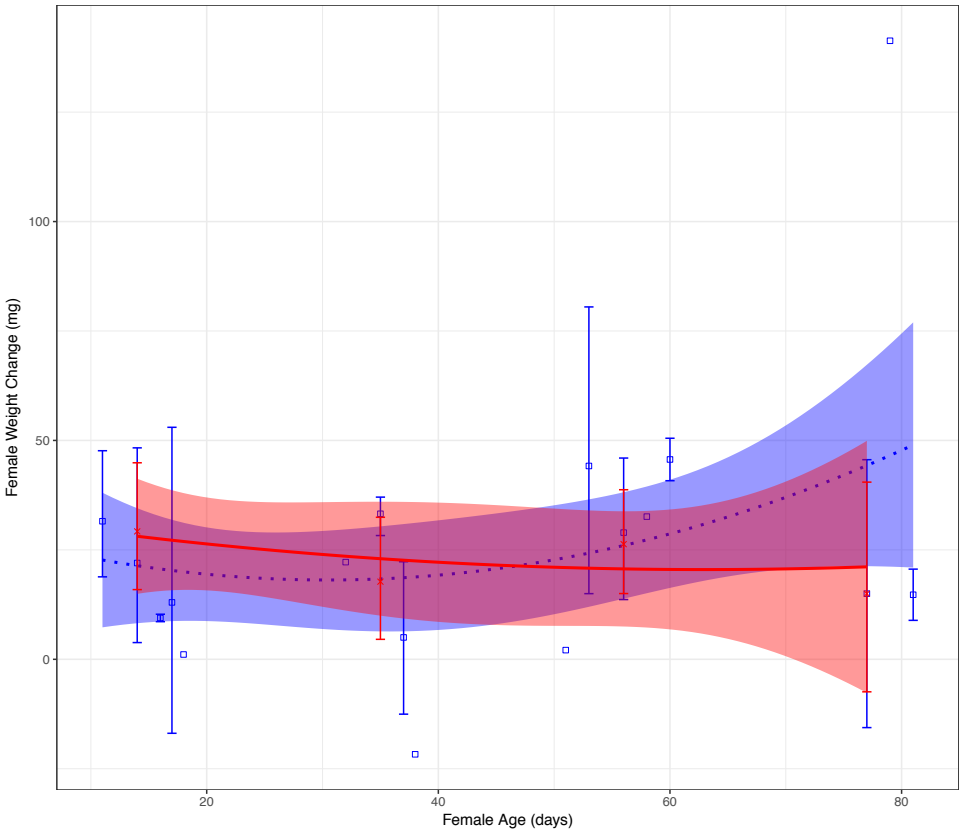

Fig. S2.5(b) Quadratic fit of carer weight change to the age of mating (blue dotted line with open squares) and egg-producers (red solid line with crosses). Smoothed lines signify predicted values from linear or quadratic models. The coloured areas show calculated 95% confidence intervals. Error bars represent standard errors.

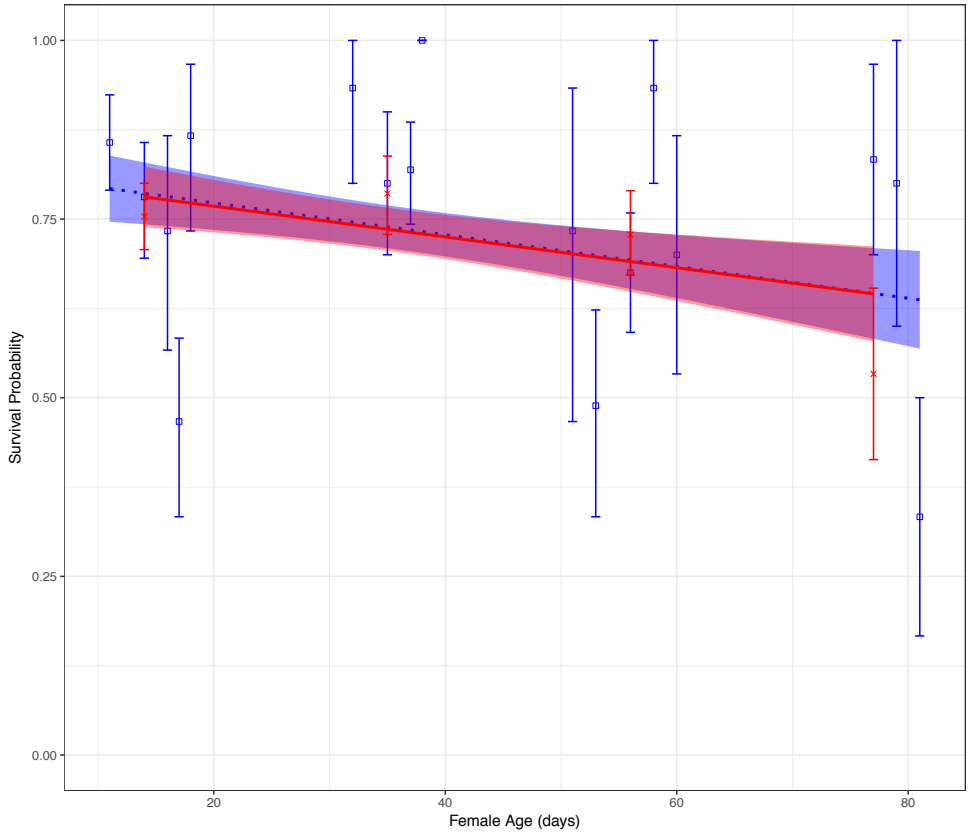

Fig. S2.6(a) Linear fit of survival probability of larvae reaching dispersal to the age of carers (blue dotted line with open squares) and egg-producers (red solid line with crosses). Smoothed lines signify predicted values from linear or quadratic models. The coloured areas show calculated 95% confidence intervals. Error bars represent standard errors.

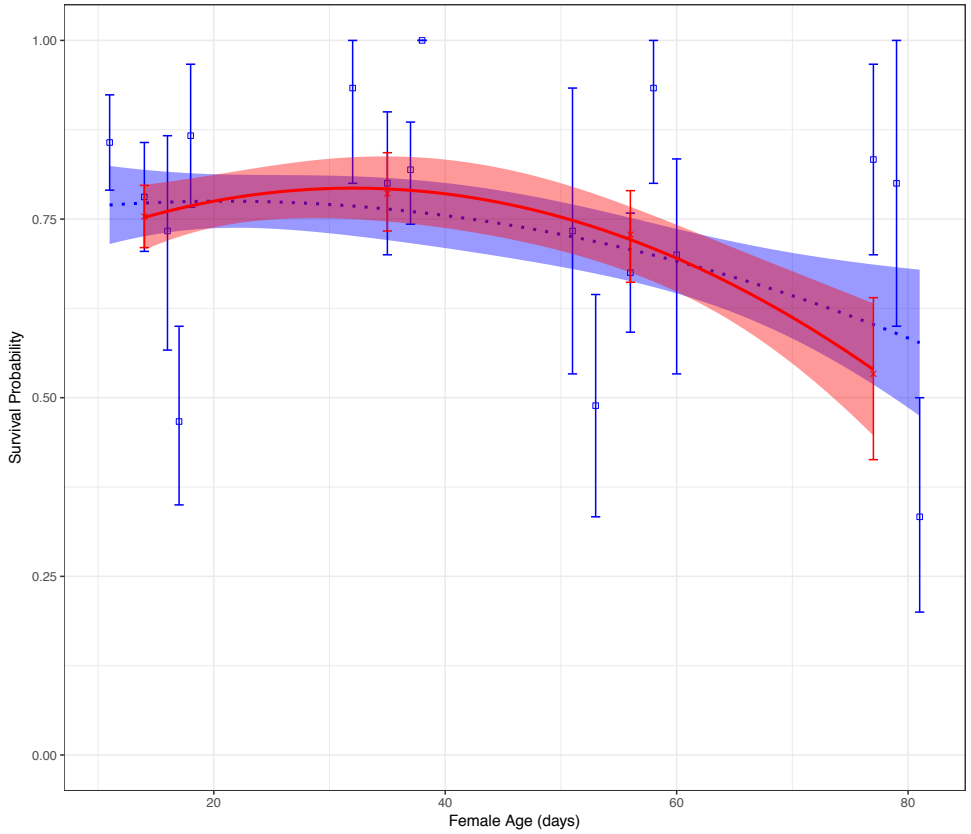

Fig. S2.6(b) Quadratic fit of survival probability of larvae reaching dispersal to the age of carers (blue dotted line with open squares) and egg-producers (red solid line with crosses). Smoothed lines signify predicted values from linear or quadratic models. The coloured areas show calculated 95% confidence intervals. Error bars represent standard errors.

Supplementary material from: "Evaluating the diversity of maternal age effects upon neonatal survival across animal species"

Table S3.1 Search strings used for database search

Terms Relating to Maternal Age	Terms Relating to Offspring Production	Terms Relating to Neonatal Survival and Maternal Care
Age*	Fecundity	Viability
Senescence	Offspring*	Fledgling*
Maternal Age	Seed*	Survival
Parental Age	Egg*	Mortality
-	Fertility	Offspring to Adult Success
-	Reproduct*	Parental*

Note - Often the additional search string of life histor* was added which removed superfluous search results. Search strings between columns were separated with the "and" term, within column with an "or" term. An asterisk next to a string allows you to search for words that start with the same letters.

Table S3.2. Parameter constraints for each of the tested models

Model	C	A/γ	B/α	β
Gompertz	1	*Unconstrained*	0-Inf	*NA*
Gompertz-Makeham	0-1	*Unconstrained*	0-Inf	*NA*
Weibull	0	*Unconstrained*	0-Inf	0-Inf
Evolutionary	0	*Unconstrained*	0-Inf	-1

Table S3.3 An overview of published sources of data with corresponding numbers of neonates, mothers, and age classes.

Species	Author	Replicate	No. Neonates	No. Age Classes	Generation Length (*T*)
Accipiter nisus	Newton, 2002	1	739	8	3.48 Years
Acutuncus antarcticus	Tsujimoto, 2016	1	19378	155	46.75 Days
Alces alces	Ericsson, 2001	1	407	14	7.78 Years
Anoplophora glabripennis	Smith, 2002	1	2504	12	7.64 Weeks
		2	1405	12	7.19 Weeks
		3	598	12	6.32 Weeks
Anser caerulescens	Rockwell, 1993	1	2179	9	6.64 Years
Aphelinus gossypii	Perng, 2002	1	9872	31	11.21 Days
		2	7464	31	9.75 Days
Aphidius transcaspicus	Latham, 2010	1	8274	10	3.90 Days
Branta sandvicensis	Woog, 2002	1	1171	14	5.34 Years
Calanus sinicus	Lin, 2015	1	645	9	7.12 Days

		2	124	9	3.63 Days
		3	202	9	3.30 Days
Canis lupus	Stahler, 2013	1	579	8	4.46 Years
Catharacta skua	Ratcliffe, 1998	1	1372	20	12.76 Years
		2	1407	24	13.10 Years
		3	1381	21	12.99 Years
Cheilomenes sexmaculata	Omkar, 2006	1	60780	9	32.36 Days
Chilo suppressalis	Kanno, 1975	1	10038	7	2.18 Days
Conchyloctenia hybrida	Ghebremariam, 2014	1	2949	5	3.95 Months
Drosophila littoralis	Pekkala, 2011	1	127042	59	45.31 Days
Drosophila melanogaster	Fowler, 1989	1	42583	17	13.81 Days
		2	49136	18	15.04 Days
Drosophila mercatorum	Kramer, 2001	1	16106	16	12.29 Days
		2	13437	15	11.12 Days
		3	19246	16	14.41 Days
		4	16036	15	11.81 Days

Elephas maximus	Robinson, 2012	1	1111	9	23.41 Years
Falco columbarius	Espie, 2000	1	205	6	2.99 Years
Ficedula albicollis	Gustafsson, 1990	1	4914	4	1.63 Years
Ficedula hypoleuca	Potti, 2013	1	2865	4	2.09 Years
		2	1264	4	2.76 Years
Galba cubensis	Gutierrez, 2000	1	45098	24	9.67 Weeks
		2	20528	16	8.32 Weeks
Gorilla beringei	Robbins, 2006	1	208	8	16.77 Years
Helicoverpa armigera	Jha, 2014	1	12737	23	39.02 Days
	Jha, 2012	1	13176	21	46.25 Days
		2	5650	18	47.21 Days
	Jha, 2012	1	6419	28	55.96 Days
	Liu, 2017	1	40627	14	27.7 Days
		2	26007	10	27.93 Days
		3	19558	10	28.27 Days

Hirundo rustica	Balbontin, 2012	1	3629	6	1.88 Years
		2	9391	6	1.5 Years
		3	3601	5	1.84 Years
		4	9355	5	1.48 Years
		5	3474	4	1.73 Years
		6	9243	4	1.44 Years
Lacerta vivipara	Richard, 2005	1	1758	5	3.09 Years
Lagopus muta japonica	Suzuki, 2013	1	302	5	2.65 Years
Larus audouinii	Oro, 2014	1	3768	23	9.00 Years
Larus californicus	Pugesek, 1983	1	624	15	9.39 Years
Larus heermanni	Vieyra, 2009	1	2163	10	6.84 Years
Larus novaehollandiae scopulinus	Mills, 1973	1	904	9	5.93 Years
Lemur catta	Parga, 2005	1	116	13	6.31 Years
Lobesia botrana	Moreau, 2016	1	26603	7	2.22 Days
Lucilia cuprina	Readshaw, 1983	1	64632	26	13.66 Days
		2	3495	8	10.49 Days
		3	3218	9	10.70 Days

		4	51048	21	12.17 Days
Macaca mulatta	Gagliardi, 2007	1	10644	22	10.31 Years
Milvus migrans	Blas, 2009	1	897	10	5.99 Years
Nezara viridula	Kiritani, 1963	1	4012	10	47.77 Days
	Kiritani, 1967	1	7190	8	39.24 Days
Orius niger	Baniameri, 2005	1	1185	27	28.57 Days
		2	702	17	22.37 Days
		3	881	14	21.08 Days
Ovis aries	Hayward, 2013	1	2639	12	5.10 Years
	Hayward, 2015	1	2235	8	5.68 Years
Panthera leo	Packer, 1998	1	2810	13	6.76 Years
Panthera pardus	Balme, 2013	1	309	14	7.62 Years
Papio anubis	Packer, 1998	1	670	19	11.16 Years
Parus major	Bouwhuis, 2009	1	59329	9	1.89 Years
	Bouwhuis, 2010	1	21664	7	1.87 Years
		2	21664	7	1.87 Years
		3	21664	7	1.87 Years

	Perrins, 2008	1	10358	7	1.89 Years
Physa acuta	Auld, 2014	1	11091	7	14.86 Weeks
Podisus nigrispinus	DeCastro, 2015	1	1592	18	20.49 Days
		2	1503	18	21.80 Days
		3	898	16	16.64 Days
	Medeiros, 2000	1	4497	8	50.33 Days
Rangifer tarandus	Jorgensen, 2015	1	633	16	6.97 Years
Sialia mexicana	Keyser, 2004	1	1133	4	1.68 Years
Spodoptera exigua	Rogers, 1996	1	255114	9	3.87 Days
	Rogers, 1997	1	181509	9	4.37 Days
Tamiasciurus hudsonicus	Descamps, 2008	1	1745	7	2.83 Years
Tyrannus tyrannus	Murphy, 2004	1	360	5	3.32 Years
Urocitellus columbianus	Skibiel, 2009	1	261	5	3.87 Years

Note - Estimates of generation time estimate *T* is defined in subsection "Scaling maternal ages to generation time"). See accompanying literature references below.

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Fig. S3.1 A histogram of the among-replicate distribution of oldest mothers surveyed.

Showing number of treatments with the highest surveyed age class (standardized by T)

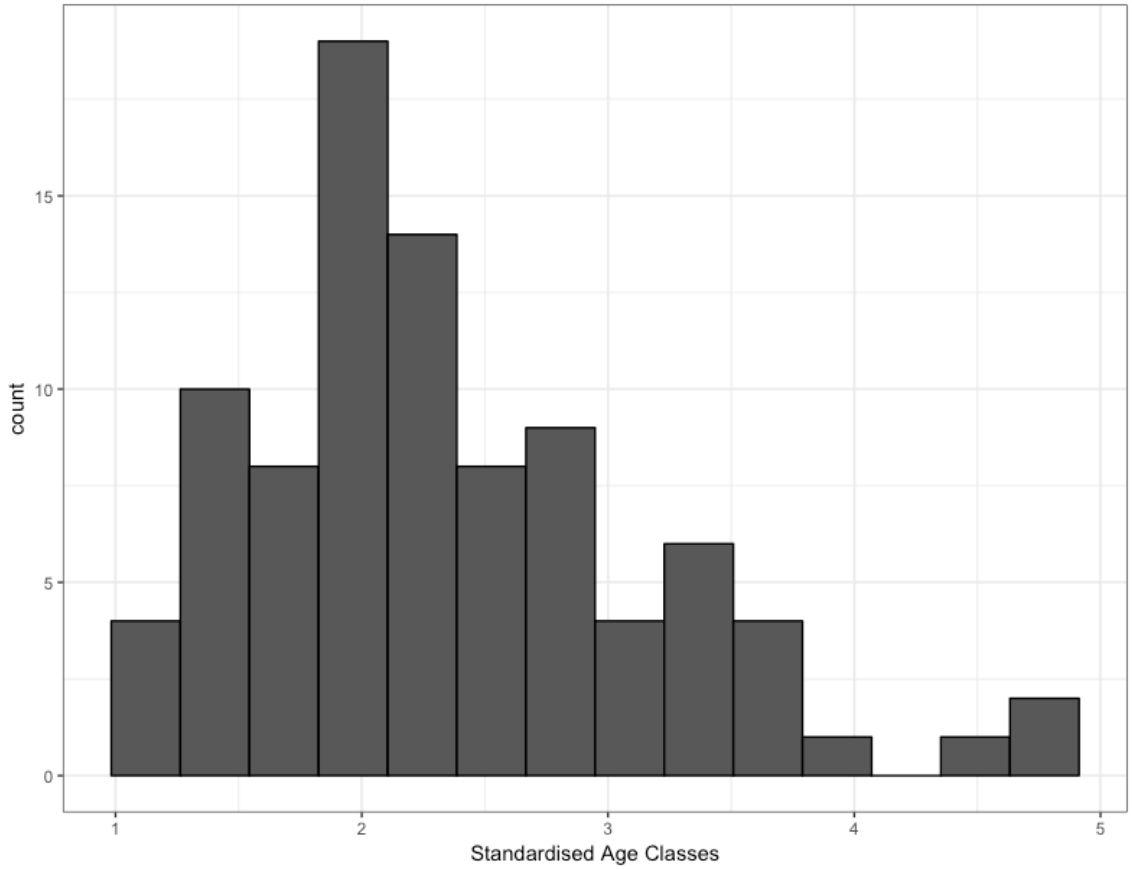


Table S3.4 Replicate-specific AICcs from age-independent, linear and quadratic GLMs

Author	Year	*Species*	Environment	AICc Null	AICc Linear	AICc Quad	Ranking Null	Ranking Linear	Ranking Quad
Woog	2002	*Branta sandvicensis*	Captive	1593.3634	1593.5409	1594.1687	1	2	3
Robinson	2012	*Elephas maximus*	Captive	1444.9014	1439.5143	1425.7172	3	2	1
Richard	2005	*Lacerta vivipara*	Captive	2436.7232	2435.1393	2367.8728	3	2	1
Parga	2005	*Lemur catta*	Captive	146.7643	147.7336	147.6298	1	3	2
Gagliardi	2007	*Macaca mulatta*	Captive	10873.8660	10872.2092	10867.7460	3	2	1
Jorgensen	2015	*Rangifer tarandus*	Captive	191.0475	186.4909	180.3298	3	2	1
Skibiel	2009	*Urocitellus columbianus*	Captive	136.1604	133.0526	133.2327	3	1	2
Tsujimoto	2016	*Acutuncus antarcticus*	Lab	6890.9428	6589.3725	6588.6927	3	2	1
Smith 1	2002	*Anoplophora glabripennis*	Lab	3211.1556	3014.3140	3013.6611	3	2	1
Smith 2	2002	*Anoplophora glabripennis*	Lab	1946.6637	1815.6013	1816.6043	3	1	2
Smith 3	2002	*Anoplophora glabripennis*	Lab	747.6781	716.7148	717.7217	3	1	2
Perng 1	2002	*Aphelinus gossypii*	Lab	4643.0203	4629.0474	4629.5189	3	1	2
Perng 2	2002	*Aphelinus gossypii*	Lab	2867.2857	2858.2194	2854.5783	3	2	1
Latham	2010	*Aphidius transcaspicus*	Lab	10903.9945	10867.8010	10826.3184	3	2	1
Lin 1	2015	*Calanus sinicus*	Lab	704.3774	705.3855	704.2217	2	3	1
Lin 2	2015	*Calanus sinicus*	Lab	164.6022	156.2942	156.2232	3	2	1
Lin 3	2015	*Calanus sinicus*	Lab	189.3666	189.4953	190.4027	1	2	3
Omkar	2006	*Cheilomenes sexmaculata*	Lab	51304.6568	51178.4268	51056.8927	3	2	1
Kanno	1975	*Chilo suppressalis*	Lab	13810.1601	13592.2816	13561.4973	3	2	1
Ghebremariam	2014	*Conchyloctenia hybrida*	Lab	3865.6257	3784.5945	3784.5607	3	2	1
Pekkala	2011	*Drosophila littoralis*	Lab	168101.0602	157087.6914	156708.3777	3	2	1
Fowler 1	1989	*Drosophila melanogaster*	Lab	55100.6541	52257.8162	51943.4842	3	2	1

Fowler 2	1989	*Drosophila melanogaster*	Lab	64775.6846	60936.5746	60932.6746	3	2	1
Kramer 1	2001	*Drosophila mercatorum*	Lab	2718.7883	2680.5115	2658.3958	3	2	1
Kramer 2	2001	*Drosophila mercatorum*	Lab	4420.8850	4361.8733	4362.7566	3	1	2
Kramer 3	2001	*Drosophila mercatorum*	Lab	6215.5602	6160.7976	6133.2737	3	2	1
Kramer 4	2001	*Drosophila mercatorum*	Lab	5139.5292	5118.0331	5100.5194	3	2	1
Gutierrez 1	2000	*Galba cubensis*	Lab	58208.8089	58193.1844	58090.6936	3	2	1
Gutierrez 2	2000	*Galba cubensis*	Lab	26606.4913	26517.8412	26503.6870	3	2	1
Jha 12	2012	*Helicoverpa armigera*	Lab	13098.4394	11660.0627	11028.7317	3	2	1
Jha 12 1	2012	*Helicoverpa armigera*	Lab	17517.0537	17422.6709	16340.3133	3	2	1
Jha 12 2	2012	*Helicoverpa armigera*	Lab	7584.2978	7493.5768	6928.0821	3	2	1
Jha 14	2014	*Helicoverpa armigera*	Lab	8612.1432	8349.0725	8261.6314	3	2	1
Liu 1	2017	*Helicoverpa armigera*	Lab	56019.2561	54466.1288	54311.1027	3	2	1
Liu 2	2017	*Helicoverpa armigera*	Lab	34860.2407	34184.2897	33571.1884	3	2	1
Liu 3	2017	*Helicoverpa armigera*	Lab	26353.4871	26059.3375	26060.2088	3	1	2
Moreau	2016	*Lobesia botrana*	Lab	20738.3501	20062.0618	20034.3189	3	2	1
Readshaw 1	1983	*Lucilia cuprina*	Lab	52562.8377	50177.6218	49510.7446	3	2	1
Readshaw 2	1983	*Lucilia cuprina*	Lab	4566.0117	4564.1828	4560.4461	3	2	1
Readshaw 3	1983	*Lucilia cuprina*	Lab	4229.5415	4111.7082	3947.6853	3	2	1
Readshaw 4	1983	*Lucilia cuprina*	Lab	62714.5899	60842.6012	60822.6177	3	2	1
Kiritani	1963	*Nezara viridula*	Lab	4562.0147	2632.9925	2513.0226	3	2	1
Kiritani	1967	*Nezara viridula*	Lab	4414.4875	4205.9028	4200.5766	3	2	1
Baniameri 1	2005	*Orius niger*	Lab	1218.4780	1207.8941	1207.9862	3	1	2
Baniameri 2	2005	*Orius niger*	Lab	442.1956	441.3282	442.3416	2	1	3
Baniameri 3	2005	*Orius niger*	Lab	610.9408	610.6231	610.6065	3	2	1
Auld	2014	*Physa acuta*	Lab	15350.3408	12415.2960	11902.8598	3	2	1

DeCastro 1	2015	*Podisus nigrispinus*	Lab	885.8446	871.8182	856.2344	3	2	1
DeCastro 2	2015	*Podisus nigrispinus*	Lab	831.9124	832.2184	831.7531	2	3	1
DeCastro 3	2015	*Podisus nigrispinus*	Lab	580.3300	563.4228	564.2124	3	1	2
Medeiros	2000	*Podisus nigrispinus*	Lab	5686.6477	5503.4838	5382.8149	3	2	1
Rogers	1996	*Spodoptera exigua*	Lab	310529.0759	278789.0120	278383.7403	3	2	1
Rogers	1997	*Spodoptera exigua*	Lab	170093.0981	161267.9347	161244.5009	3	2	1
Newton	2002	*Accipiter nisus*	Natural	958.5921	953.6752	953.3961	3	2	1
Ericsson	2001	*Alces alces*	Natural	563.1476	560.7937	560.6263	3	2	1
Rockwell	1993	*Anser caerulescens*	Natural	2980.6763	2981.3382	2966.3600	2	3	1
Stahler	2013	*Canis lupus*	Natural	793.4430	792.6744	793.1966	3	1	2
Ratcliffe 1	1998	*Catharacta skua*	Natural	1077.7512	1074.9105	1075.4314	3	1	2
Ratcliffe 2	1998	*Catharacta skua*	Natural	1940.4423	1777.3882	1770.4791	3	2	1
Ratcliffe 3	1998	*Catharacta skua*	Natural	1885.1163	1864.9802	1849.2480	3	2	1
Espie	2000	*Falco columbarius*	Natural	91.4174	91.5444	92.4251	1	2	3
Gustafsson	1990	*Ficedula albicollis*	Natural	4517.1065	4383.4732	4384.1532	3	1	2
Potti 1	2013	*Ficedula hypoleuca*	Natural	3778.9445	3765.3969	3765.4019	3	1	2
Potti 2	2013	*Ficedula hypoleuca*	Natural	1687.0034	1686.5718	1687.3463	2	1	3
Robbins	2006	*Gorilla beringei*	Natural	236.1223	236.4000	235.2902	2	3	1
Balbontin 1	2012	*Hirundo rustica*	Natural	5012.9304	5004.9605	5001.9500	3	2	1
Balbontin 2	2012	*Hirundo rustica*	Natural	10660.0456	10660.4953	10660.3207	1	3	2
Balbontin 3	2012	*Hirundo rustica*	Natural	4849.9355	4849.7404	4849.6575	3	2	1
Balbontin 4	2012	*Hirundo rustica*	Natural	9953.4950	9927.9125	9925.6737	3	2	1
Balbontin 5	2012	*Hirundo rustica*	Natural	4590.6274	4590.0076	4590.6201	3	1	2
Balbontin 6	2012	*Hirundo rustica*	Natural	9412.7644	9385.9805	9384.9892	3	2	1
Suzuki	2013	*Lagopus muta japonica*	Natural	172.4617	173.1483	156.0178	2	3	1

Oro	2014	*Larus audouinii*	Natural	4981.4369	4981.6301	4962.6685	2	3	1
Pugesek	1983	*Larus californicus*	Natural	864.4130	830.7700	831.6527	3	1	2
Vieyra	2009	*Larus heermanni*	Natural	2899.5460	2892.8187	2892.1113	3	2	1
Mills	1973	*Larus novaehollandiae scopulinus*	Natural	1244.2395	1245.1839	1244.7300	1	3	2
Blas	2009	*Milvus migrans*	Natural	1124.5480	1100.2469	1099.6562	3	2	1
Hayward	2013	*Ovis aries*	Natural	2573.7809	2491.2646	2356.0350	3	2	1
Hayward	2015	*Ovis aries*	Natural	2891.2926	2857.9563	2858.3466	3	1	2
Packer	1998	*Panthera leo*	Natural	3543.5013	3535.1063	3534.7648	3	2	1
Balme	2013	*Panthera pardus*	Natural	405.8073	403.8406	401.8199	3	2	1
Packer	1998	*Papio anubis*	Natural	809.9126	809.6393	796.8836	3	2	1
Bouwhuis	2009	*Parus major*	Natural	58666.2454	58574.7102	58522.1022	3	2	1
Bouwhuis 1	2010	*Parus major*	Natural	12664.7422	12665.7163	12665.8927	1	2	3
Bouwhuis 2	2010	*Parus major*	Natural	12579.2936	12577.7284	12567.4272	3	2	1
Bouwhuis 3	2010	*Parus major*	Natural	12483.8985	12480.7981	12481.1013	3	1	2
Perrins	2008	*Parus major*	Natural	7533.3713	7500.7563	7493.7899	3	2	1
Keyser	2004	*Sialia mexicana*	Natural	1473.7501	1472.1960	1464.0287	3	2	1
Descamps	2008	*Tamiasciurus hudsonicus*	Natural	1975.0852	1974.4706	1946.0317	3	2	1
Murphy	2004	*Tyrannus tyrannus*	Natural	489.6918	477.9689	477.4897	3	2	1

Table S3.5 Constrained model parameters, showing species, paper, and parameter values for the demographic and evolutionary models

Species	Paper	Replicate	Gompertz			Gompertz-Makeham				Weibull				Evolutionary		
			LogL	A	B	*LogL*	C	A	B	*LogL*	γ	α	β	*LogL*	γ	α
Accipiter nisus	Newton, 2002	-	-479.29	-0.84	0.00	-479.29	0.65	-4.57	0.00	-479.29	-0.65	0.00	0.99	-479.29	-0.65	0.00
Acuntuncus antarcticus	Tsujimoto, 2016	-	-3293.93	-4.12	0.84	-3293.78	1.00	-4.29	0.91	-3287.69	-0.98	0.01	2.42	-3348.02	-0.98	0.00
Alces alces	Ericsson, 2001	-	-279.84	-0.62	0.33	-278.87	0.49	-21.66	11.85	-279.02	-0.49	0.00	11.27	-280.30	-0.53	0.01
Anoplophora glabripennis	Smith, 2002	1	-1605.58	-0.88	0.00	-1605.58	0.66	-8.58	0.00	-1605.58	-0.66	0.00	1.01	-1605.58	-0.66	0.00
		2	-973.33	-0.33	0.00	-973.33	0.49	-12.72	0.00	-973.33	-0.49	0.00	1.19	-973.33	-0.49	0.00
		3	-373.84	-0.96	0.00	-373.84	0.68	-8.50	0.00	-373.84	-0.68	0.00	1.01	-373.84	-0.68	0.00
Anser caerulescens	Rockwell, 1993	-	-1490.34	-0.57	0.00	-1489.23	0.58	-17.87	10.24	-1490.34	-0.57	0.00	1.01	-1489.89	-0.58	0.00
Aphelinus gossypii	Perng, 2002	1	-2313.98	-3.00	0.25	-2313.77	0.96	-4.68	0.74	-2313.73	-0.95	0.01	2.16	-2320.27	-0.94	0.00
		2	-1428.50	-3.30	0.27	-1427.36	0.96	-5.82	1.12	-1426.40	-0.96	0.00	2.91	-1433.64	-0.95	0.00
Aphidius transcaspicus	Latham, 2010	-	-5432.69	-0.98	0.20	-5417.70	0.65	-6.71	2.27	-5417.61	-0.65	0.00	4.81	-5419.66	-0.66	0.01
Branta sandvicensis	Woog, 2002	-	-796.68	-0.14	0.00	-796.43	0.42	-113.18	40.07	-796.68	-0.42	0.00	1.03	-796.68	-0.42	0.00
Calanus sinicus	Lin, 2015	1	-352.18	-1.32	0.01	-352.18	1.00	-1.32	0.01	-352.11	-0.78	0.02	0.26	-352.19	-0.76	0.00
		2	-77.78	-1.09	0.30	-77.78	1.00	-1.09	0.30	-75.09	-0.81	0.48	0.22	-79.66	-0.74	0.02
		3	-94.21	-1.78	0.14	-94.05	0.83	-13.50	2.64	-94.16	-0.84	0.01	1.86	-94.01	-0.84	0.00
Canis lupus	Stahler, 2013	-	-395.81	-0.77	0.22	-395.51	0.59	-6.23	2.56	-395.50	-0.58	0.01	4.78	-395.77	-0.60	0.01
Catharacta skua	Ratcliffe, 1998	1	-538.87	0.70	0.00	-538.87	0.13	-76.13	35.84	-538.87	-0.13	0.00	0.81	-538.75	-0.14	0.00

Species	Paper	Replicate	Gompertz			Gompertz-Makeham				Weibull				Evolutionary		
			LogL	A	B	*LogL*	C	A	B	*LogL*	γ	α	β	*LogL*	γ	α
		2	-970.22	-0.25	0.00	-970.22	0.55	-1.69	0.00	-970.22	-0.46	0.00	1.01	-970.22	-0.46	0.00
		3	-942.56	-0.58	0.00	-942.56	0.57	-7.57	0.00	-942.56	-0.57	0.00	1.11	-942.56	-0.57	0.00
Cheilomenes sexmaculata	Omkar, 2006	-	-25652.33	-1.82	0.00	-25652.33	0.85	-12.52	0.00	-25652.33	-0.85	0.00	1.04	-25652.33	-0.85	0.00
Chilo suppressalis	Kanno, 1975	-	-6792.75	-0.86	0.34	-6783.54	0.65	-2.92	0.97	-6778.59	-0.60	0.05	2.74	-6816.42	-0.61	0.02
Conchyloctenia hybrida	Ghebremariam, 2014	-	-1891.36	-2.13	1.32	-1891.13	0.85	-3.35	2.03	-1891.65	-0.78	0.18	3.05	-1932.81	-0.64	0.00
Drosophila littoralis	Pekkala, 2011	-	-78415.56	-1.75	0.94	-78355.27	0.87	-2.53	1.29	-78367.52	-0.77	0.16	2.52	-81215.06	-0.80	0.00
Drosophila melanogaster	Fowler, 1989	1	-26065.44	-1.79	0.89	-25900.72	0.75	-5.49	2.74	-25951.82	-0.73	0.02	5.53	-25968.88	-0.97	0.03
		2	-30487.24	-1.67	0.85	-30487.24	1.00	-1.67	0.85	-30461.11	-0.84	0.28	1.54	-31597.10	-0.79	0.01
Drosophila mercatorum	Kramer, 2001	1	-1340.55	1.26	0.17	-1340.55	1.00	1.26	0.17	-1326.14	-203.36	9.67	0.09	-1358.26	-0.02	0.00
		2	-2180.40	0.92	0.27	-2180.40	0.82	0.84	0.29	-2180.06	-0.07	0.60	1.49	-2207.91	-0.04	0.01
		3	-3080.77	1.04	0.15	-3080.77	1.00	1.04	0.15	-3067.39	-173.25	8.56	0.07	-3107.78	-0.04	0.00
		4	-2558.86	1.10	0.10	-2558.86	1.00	1.10	0.10	-2553.70	-194.82	8.64	0.06	-2568.25	-0.04	0.00
Elephas maximus	Robinson, 2012	1	-719.03	-1.21	0.38	-710.75	0.67	-12.41	6.53	-710.57	-0.67	0.00	10.75	-712.33	-0.71	0.01
Falco columbarius	Espie, 2000	1	-45.70	1.04	0.00	-45.70	0.06	-8.57	0.00	-45.70	-0.06	0.00	1.01	-45.70	-0.06	0.00
Ficedula albicollis	Gustafsson, 1990	1	-2258.55	-1.67	0.00	-2258.55	0.83	-10.64	0.00	-2258.55	-0.83	0.00	1.01	-2258.55	-0.83	0.00
Ficedula hypoleuca	Potti, 2013	1	-1889.47	-0.77	0.00	-1889.47	0.63	-15.68	0.00	-1889.47	-0.63	0.00	0.99	-1889.47	-0.63	0.00
		2	-842.79	-0.89	0.18	-842.79	1.00	-0.89	0.18	-842.55	-54.57	4.49	0.02	-843.50	-0.61	0.00

Species	Paper	Replicate	Gompertz			Gompertz-Makeham				Weibull				Evolutionary		
			LogL	A	B	*LogL*	C	A	B	*LogL*	γ	α	β	*LogL*	γ	α
Galba cubensis	Gutierrez, 2000	1	-29096.31	-0.92	0.07	-29096.31	1.00	-0.92	0.07	-29075.65	-31.93	3.90	0.01	-29104.11	-0.65	0.00
		2	-13259.13	-1.13	0.29	-13259.13	1.00	-1.13	0.29	-13248.58	-32.29	3.92	0.03	-13303.25	-0.65	0.00
Gorilla beringei	Robbins, 2006	1	-117.65	-1.51	0.28	-117.33	0.78	-5.56	2.04	-117.21	-0.77	0.02	3.69	-117.80	-0.77	0.00
Helicoverpa armigera	Jha, 2012	1	-5762.97	-3.31	3.86	-5567.14	0.36	-16.47	15.58	-5563.06	-0.36	0.40	16.65	-6154.36	-0.72	0.06
	Jha, 2012	1	-8702.68	-1.47	1.43	-8273.40	0.44	-40.44	35.57	-8270.50	-0.44	0.01	40.14	-8177.07	-0.82	0.04
		2	-3739.65	-2.25	2.17	-3622.01	0.45	-28.08	24.23	-3620.63	-0.45	0.02	27.45	-3570.19	-0.68	0.04
	Jha, 2014	1	-4166.24	-2.98	2.91	-4129.84	0.49	-14.54	12.47	-4129.83	-0.48	0.11	14.46	-4193.32	-0.56	0.02
	Liu, 2017	1	-27205.49	-4.22	3.72	-27167.23	0.73	-8.38	7.06	-27158.79	-0.70	0.21	8.83	-27820.36	-0.58	0.01
		2	-17065.50	-2.91	2.84	-16865.70	0.46	-19.53	16.81	-16862.00	-0.45	0.06	19.31	-16767.96	-0.61	0.05
		3	-13029.36	-2.59	2.49	-13029.36	1.00	-2.59	2.49	-13028.87	-0.76	0.64	3.57	-13115.99	-0.44	0.01
Hirundo rustica	Balbontin, 2012	1	-2506.46	-0.47	0.00	-2506.46	0.54	-8.25	0.00	-2506.46	-0.54	0.00	1.01	-2506.46	-0.54	0.00
		2	-5330.02	-1.22	0.00	-5329.97	0.75	-18.89	3.87	-5330.02	-0.75	0.00	0.96	-5330.02	-0.75	0.00
		3	-2424.97	-0.67	0.00	-2424.97	0.60	-12.57	0.00	-2424.97	-0.60	0.00	0.99	-2424.97	-0.60	0.00
		4	-4976.75	-1.37	0.00	-4976.75	0.83	-2.71	0.00	-4976.75	-0.78	0.00	0.99	-4976.75	-0.78	0.00
		5	-2295.31	-0.76	0.00	-2295.31	0.63	-8.80	0.00	-2295.31	-0.63	0.00	0.99	-2295.31	-0.63	0.00
		6	-4706.38	-1.46	0.00	-4706.38	0.79	-13.70	0.00	-4706.38	-0.79	0.00	0.31	-4706.38	-0.79	0.00
Lacerta vivipara	Richard, 2005	1	-1218.36	-0.35	0.00	-1209.28	0.51	-74.32	45.33	-1218.36	-0.49	0.00	1.18	-1189.20	-0.84	0.11

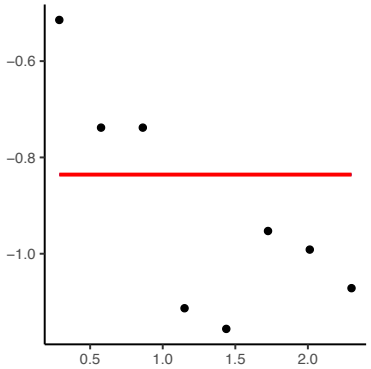
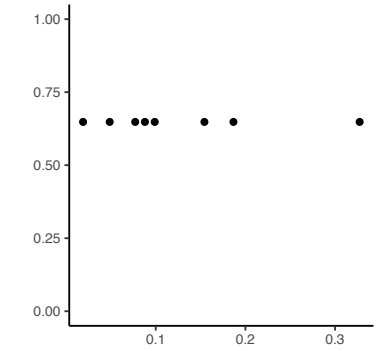
Species	Paper	Replicate	Gompertz			Gompertz-Makeham				Weibull				Evolutionary		
			LogL	A	B	*LogL*	C	A	B	*LogL*	γ	α	β	*LogL*	γ	α
Lagopus muta japonica	Suzuki, 2013	1	-86.22	-2.45	0.00	-83.65	0.93	-77.64	40.00	-84.84	-0.93	0.00	6.85	-86.22	-0.92	0.00
Larus audouinii	Oro, 2014	1	-2490.72	-0.76	0.00	-2489.88	0.63	-9.52	2.96	-2490.72	-0.63	0.00	0.99	-2490.45	-0.63	0.00
Larus californicus	Pugesek, 1983	1	-432.20	-0.41	0.00	-432.20	0.52	-4.84	0.00	-432.20	-0.52	0.00	1.01	-432.00	-0.53	0.00
Larus heermanni	Vieyra, 2009	1	-1449.77	-0.07	0.00	-1449.52	0.39	-103.37	53.57	-1449.77	-0.39	0.00	1.01	-1449.77	-0.39	0.00
Larus novaehollandiae scopulinus	Mills, 1973	1	-622.12	-0.51	0.00	-622.08	0.55	-10.55	4.58	-622.12	-0.55	0.00	1.01	-622.12	-0.55	0.00
Lemur catta	Parga, 2005	1	-73.36	-0.92	0.00	-71.96	0.68	-152.24	73.36	-73.36	-0.67	0.00	1.01	-73.11	-0.70	0.00
Lobesia botrana	Moreau, 2016	1	-10024.08	-2.70	0.67	-10017.72	0.95	-3.61	0.99	-10009.19	-0.91	0.03	2.63	-10120.60	-0.91	0.01
Lucilia cuprina	Readshaw, 1983	1	-25012.80	-3.23	1.24	-24802.10	0.92	-6.42	2.93	-24765.05	-0.91	0.02	5.04	-25688.60	-0.91	0.00
		2	-2283.01	-0.81	0.00	-2283.01	0.71	-2.24	0.00	-2283.01	-0.64	0.00	1.02	-2283.01	-0.64	0.00
		3	-2044.25	-2.99	2.18	-1987.54	0.74	-11.19	8.63	-1985.17	-0.73	0.06	11.06	-2073.39	-0.71	0.01
		4	-30435.29	-2.21	1.16	-30435.29	1.00	-2.21	1.16	-30377.17	-1.34	0.67	0.65	-31167.80	-0.72	0.00
Macaca mulatta	Gagliardi, 2007	1	-5435.58	-1.54	0.08	-5434.16	0.80	-7.67	2.35	-5433.97	-0.80	0.00	4.50	-5434.12	-0.80	0.00
Milvus migrans	Blas, 2009	1	-562.27	0.13	0.00	-562.27	0.46	-0.99	0.00	-562.27	-0.32	0.00	1.01	-562.27	-0.32	0.00
Nezara viridula	Kiritani, 1963	1	-2281.01	-1.22	0.00	-2281.01	0.79	-2.79	0.00	-2281.01	-0.74	0.00	0.96	-2281.01	-0.74	0.00
	Kiritani, 1967	1	-2100.76	-3.50	1.02	-2099.08	0.97	-4.71	1.59	-2100.32	-0.95	0.02	3.08	-2132.44	-1.04	0.02
Orius niger	Baniameri, 2005	1	-603.57	-2.55	1.09	-603.57	1.00	-2.55	1.09	-603.35	-0.96	0.19	1.43	-609.24	-0.79	0.00
		2	-220.15	-3.29	0.98	-220.15	0.98	-3.76	1.22	-220.15	-0.94	0.04	2.51	-220.81	-0.91	0.00

Species	Paper	Replicate	Gompertz			Gompertz-Makeham				Weibull				Evolutionary		
			LogL	A	B	*LogL*	C	A	B	*LogL*	γ	α	β	*LogL*	γ	α
		3	-304.79	-2.94	0.79	-304.52	0.90	-10.84	6.25	-304.50	-0.90	0.01	8.13	-304.59	-0.91	0.00
Ovis aries	Hayward, 2013	1	-1286.89	-1.55	0.00	-1286.23	0.81	-19.14	7.25	-1286.89	-0.81	0.00	1.00	-1285.46	-0.82	0.00
	Hayward, 2015	1	-1428.34	-0.57	0.62	-1428.14	0.54	-2.23	1.39	-1428.21	-0.45	0.25	2.50	-1436.06	-0.39	0.02
Panthera leo	Packer, 1998	1	-1767.10	-0.06	0.17	-1767.10	1.00	-0.06	0.17	-1766.44	-75.60	5.47	0.04	-1771.52	-0.33	0.00
Panthera pardus	Balme, 2013	1	-201.33	-0.31	0.32	-200.07	0.40	-7.73	3.96	-200.09	-0.40	0.02	6.09	-201.01	-0.44	0.02
Papio anubis	Packer, 1998	1	-404.26	-1.29	0.22	-399.60	0.73	-12.77	6.20	-399.52	-0.73	0.00	9.68	-399.03	-0.78	0.01
Parus major	Bouwhuis, 2009	1	-29285.71	-1.68	0.15	-29264.20	0.82	-5.48	1.16	-29261.41	-0.81	0.00	3.57	-29304.02	-0.81	0.00
	Bouwhuis, 2010	1	-6332.37	0.90	0.00	-6332.14	0.09	-11.83	2.92	-6332.20	-0.09	0.00	5.00	-6332.16	-0.09	0.00
		2	-6288.34	0.88	0.03	-6284.97	0.09	-5.77	1.68	-6284.46	-0.09	0.00	4.60	-6285.89	-0.09	0.00
		3	-6241.95	0.91	0.00	-6241.95	0.10	-2.00	0.00	-6241.95	-0.12	0.32	0.00	-6241.95	-0.08	0.00
	Perrins, 2008	1	-3749.49	-2.33	0.25	-3745.97	0.90	-5.53	1.16	-3746.13	-0.89	0.00	3.14	-3750.99	-0.89	0.00
Physa acuta	Auld, 2014	1	-6370.02	-2.37	2.01	-6370.02	1.00	-2.37	2.01	-6133.20	-1.21	1.01	1.61	-7675.17	-0.48	0.00
Podisus nigrispinus	DeCastro, 2015	1	-436.09	-3.22	0.69	-436.09	1.00	-3.22	0.69	-431.33	-1.05	0.13	0.57	-442.92	-0.92	0.00
		2	-415.61	-2.65	0.16	-415.61	1.00	-2.65	0.16	-415.24	-10.16	2.40	0.01	-415.95	-0.92	0.00
		3	-281.15	-3.15	0.81	-281.12	0.98	-3.53	0.95	-280.69	-0.94	0.03	2.42	-284.58	-0.93	0.00
	Medeiros, 2000	1	-2740.92	-2.75	1.79	-2665.04	0.71	-16.25	10.81	-2664.08	-0.71	0.00	15.74	-2747.35	-0.76	0.02
Rangifer tarandus	Jorgensen, 2015	1	-95.52	-3.34	0.00	-95.46	0.97	-20.78	7.72	-95.52	-0.97	0.00	1.31	-93.42	-0.97	0.00

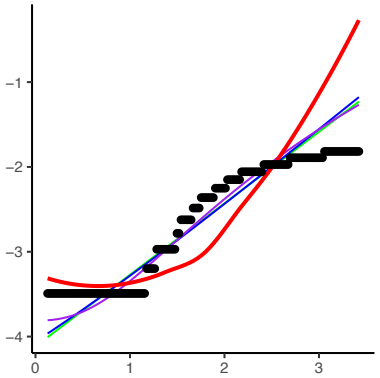
Species	Paper	Replicate	Gompertz			Gompertz-Makeham				Weibull				Evolutionary		
			LogL	A	B	*LogL*	C	A	B	*LogL*	γ	α	β	*LogL*	γ	α
Sialia mexicana	Keyser, 2004	1	-736.87	-0.83	0.00	-734.19	0.65	-57.22	23.48	-736.87	-0.65	0.00	1.01	-736.75	-0.65	0.00
Spodoptera exigua	Rogers, 1996	1	-139243.38	-0.46	0.70	-139219.72	0.82	-0.79	0.82	-139022.99	-0.48	0.48	1.97	-145774.30	-0.58	0.09
	Rogers, 1997	1	-80626.78	0.13	0.45	-80626.78	1.00	0.13	0.45	-80578.21	-0.30	0.54	1.58	-82341.82	-0.26	0.06
Tamiasciurus hudsonicus	Descamps, 2008	1	-986.68	0.24	0.08	-977.22	0.26	-78.45	36.96	-978.24	-0.27	0.00	9.55	-980.24	-0.31	0.03
Tyrannus tyrannus	Murphy, 2004	1	-238.66	-1.40	0.78	-238.66	1.00	-1.40	0.78	-237.54	-114.61	5.33	0.09	-242.46	-0.68	0.03
Urocitellus columbianus	Skibiel, 2009	1	-65.94	-3.99	1.30	-65.62	0.96	-8.21	3.94	-65.57	-0.96	0.01	5.79	-68.07	-0.93	0.00

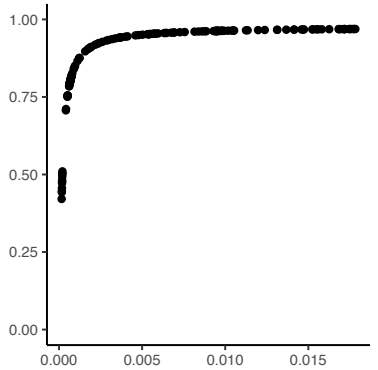
Table S3.6 Constrained model parameters, showing species, paper and model fits for the demographic and evolutionary models (if one line is shown, then the best fit is one where the slope=0 for all models).

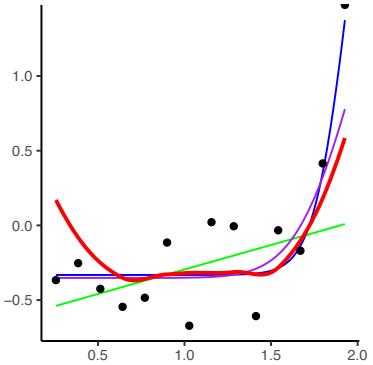
N.B. Green: Gompertz; Blue: Gompertz-Makeham; Purple: Weibull; Red: Evolutionary

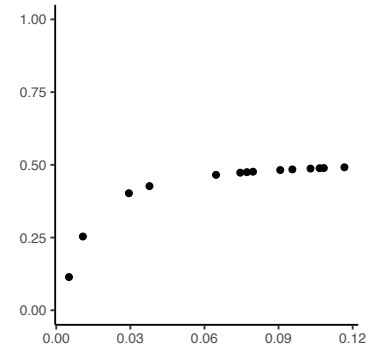
Species	Paper	Replicate	Log(-Log) Survival	Maternal Probability Distribution Function
Accipiter nisus	Newton, 2002	-		

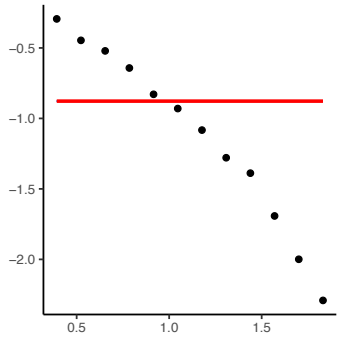
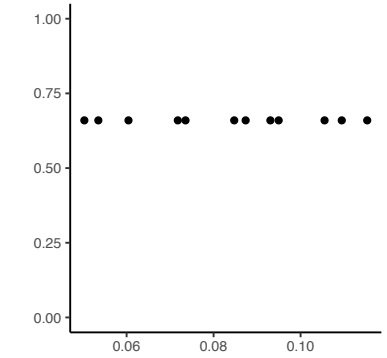
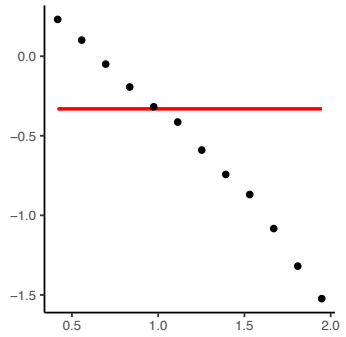
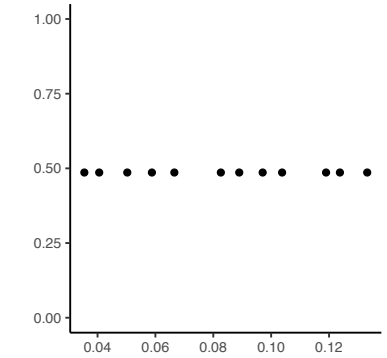
Acuntuncus antarcticus	Tsujimoto, 2016	-
Alces alces	Ericsson, 2001	-









Anoplophora glabripennis	Smith, 2002	1		
		2		

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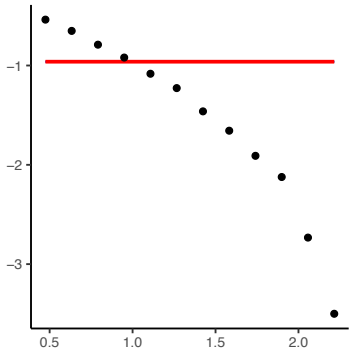
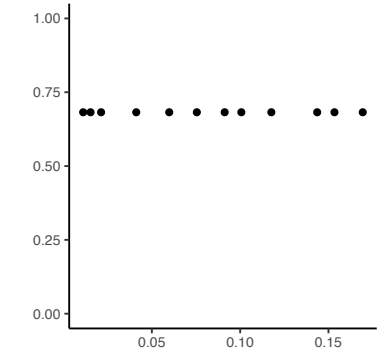
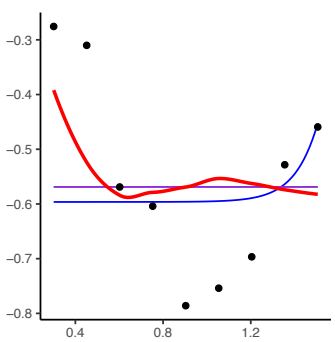
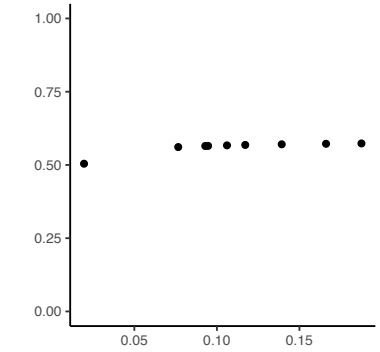
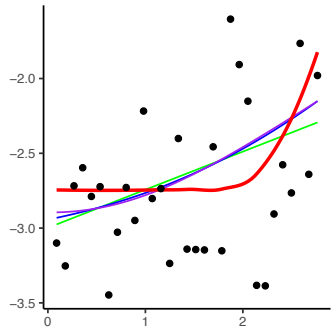
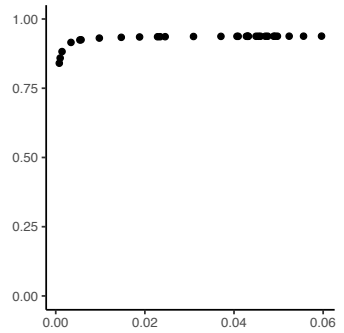
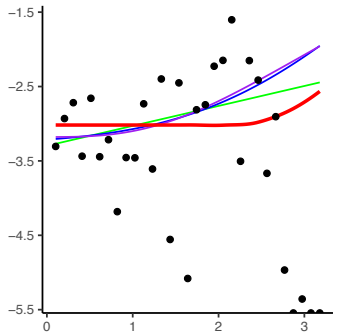
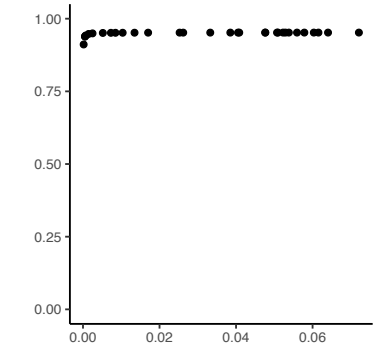
Anser caerulescens	Rockwell, 1993	-



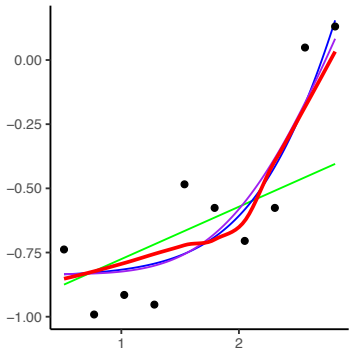


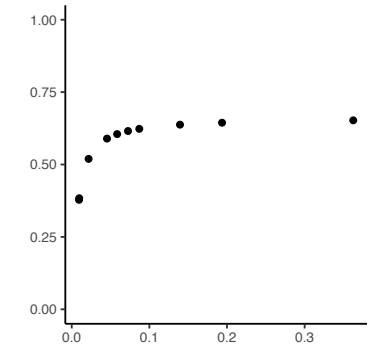


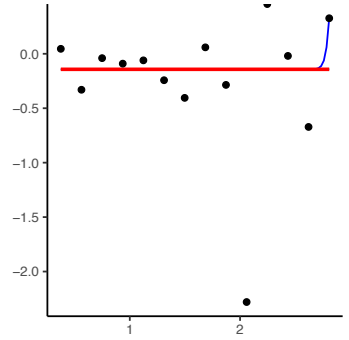


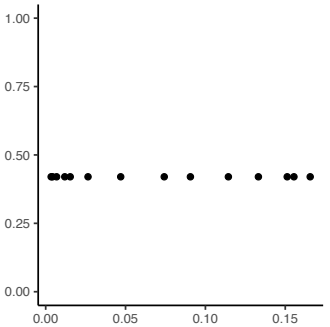
Aphelinus gossypii	Perng, 2002	1		
		2		

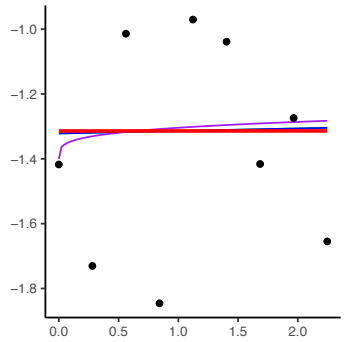
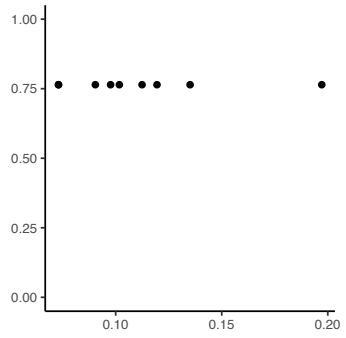
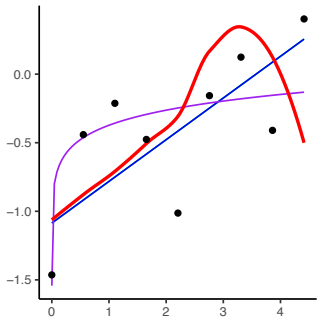
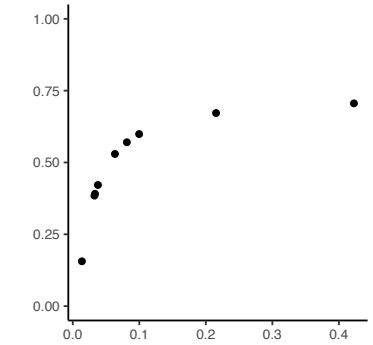
Aphidius transcaspicus	Latham, 2010	-
Branta sandvicensis	Woog, 2002	-









Calanus sinicus	Lin, 2015	1		
		2		

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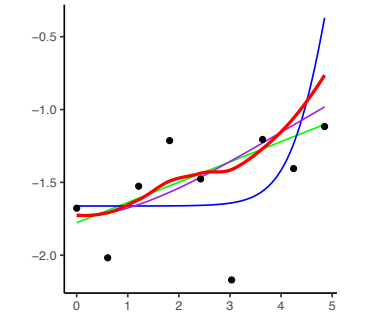
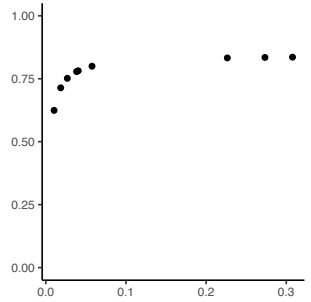
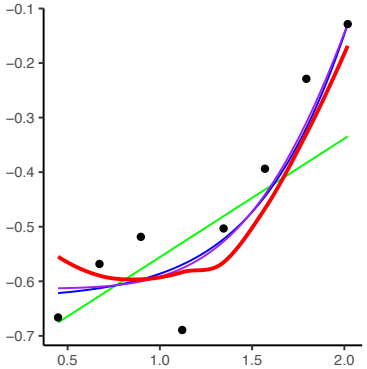
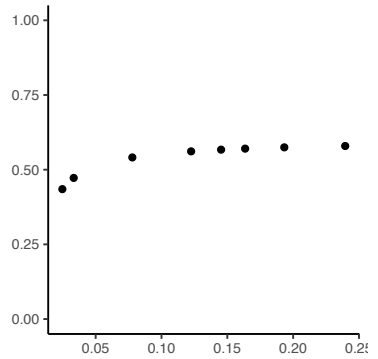
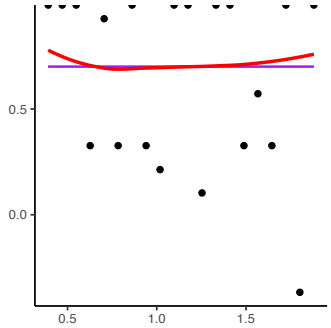
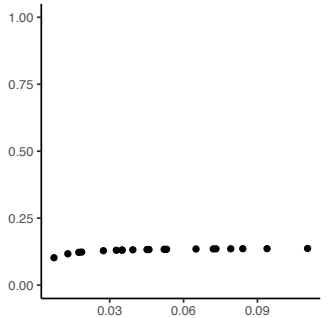
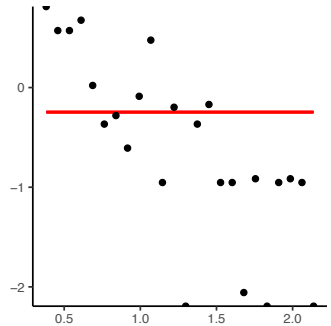
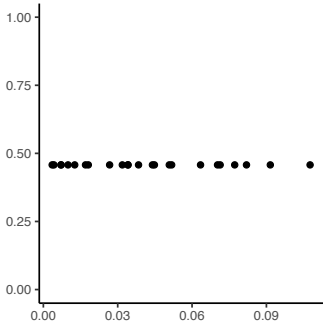
Canis lupus	Stahler, 2013	-









Catharacta skua	Ratcliffe, 1998	1		
		2		

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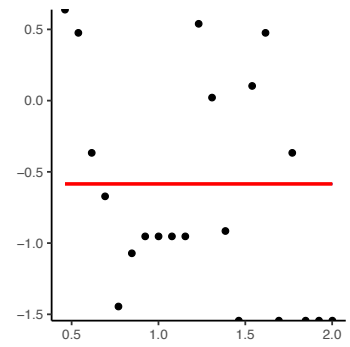
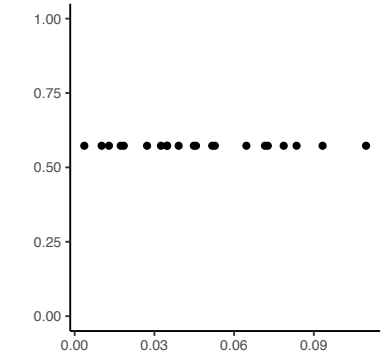
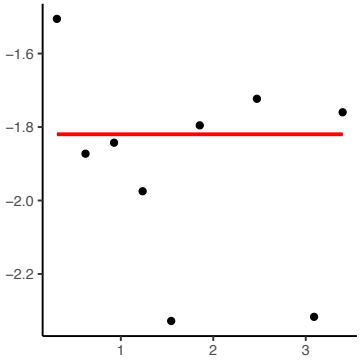
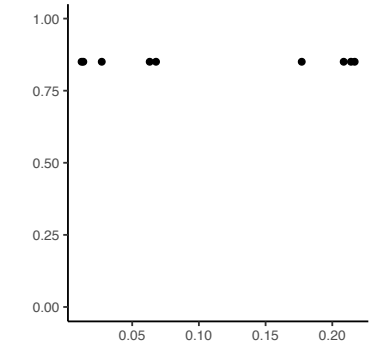
Cheilomenes sexmaculata	Omkar, 2006	-

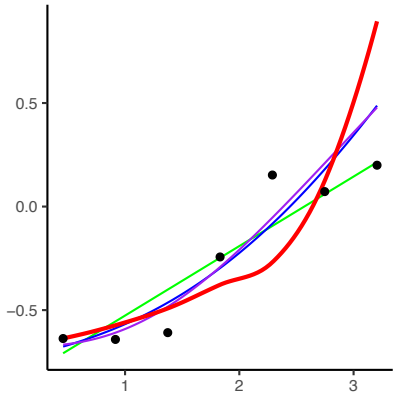


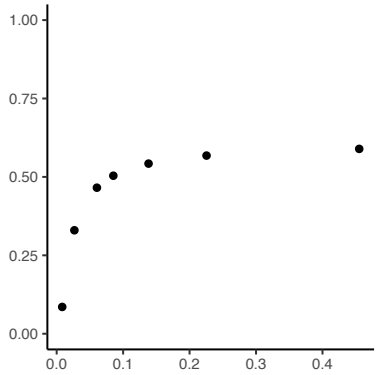


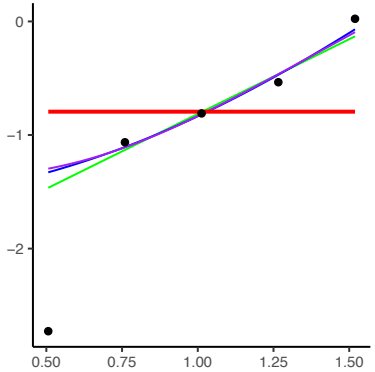


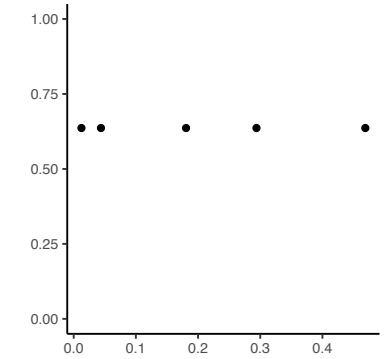


Chilo suppressalis	Kanno, 1975	-
Conchyloctenia hybrida	Ghebremariam, 2014	-

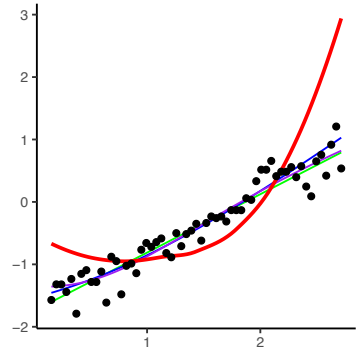


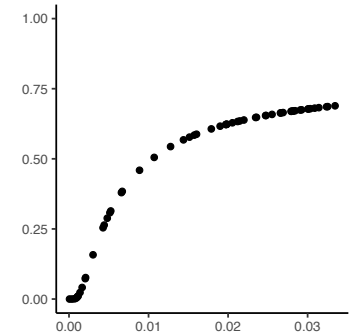


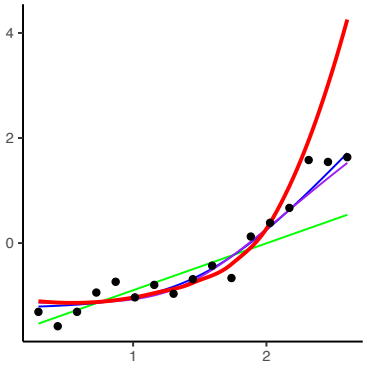


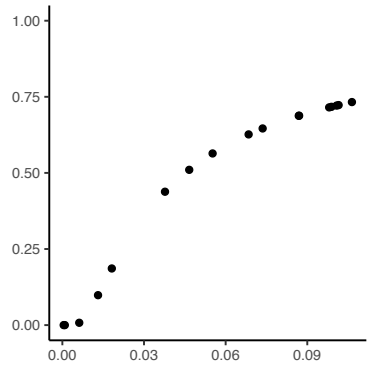


Drosophila littoralis	Pekkala, 2011	-
Drosophila melanogaster	Fowler, 1989	1









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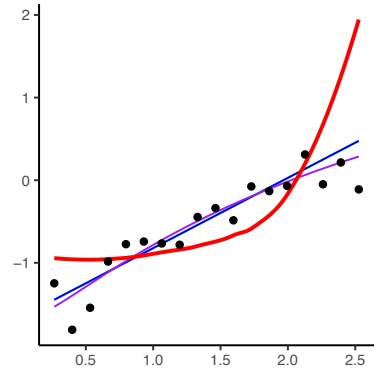
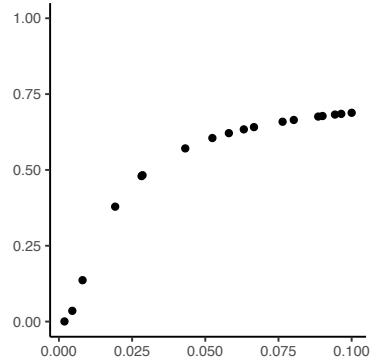
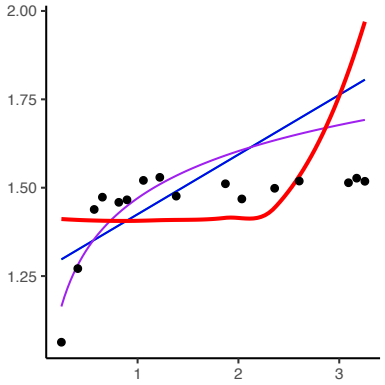
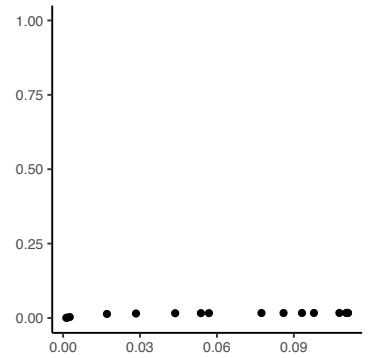
Drosophila mercatorum	Kramer, 2001	1









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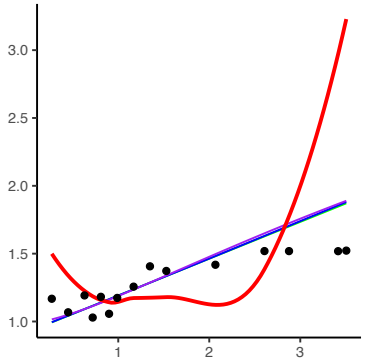
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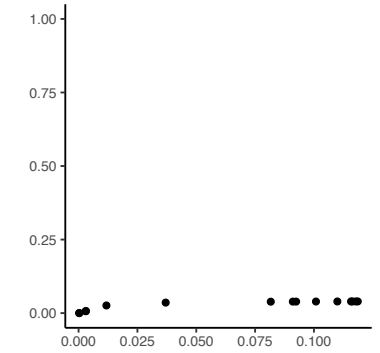
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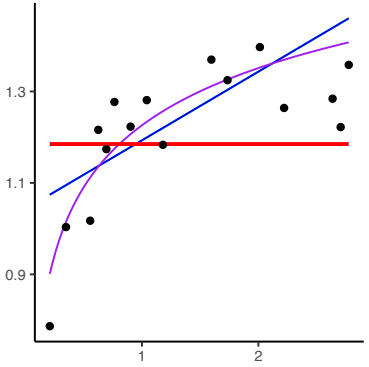
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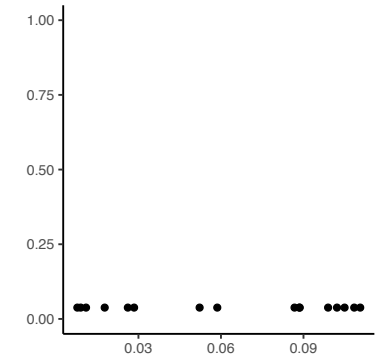
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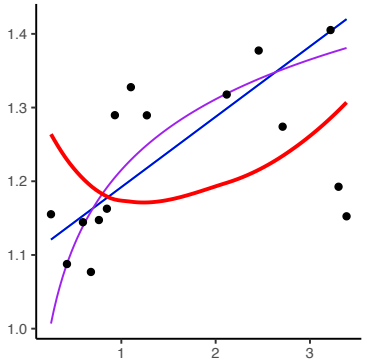
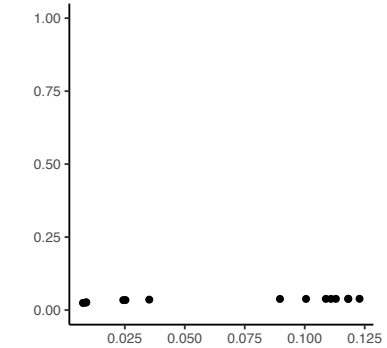
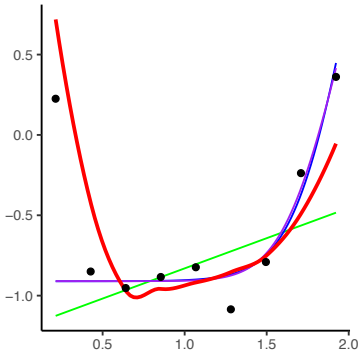
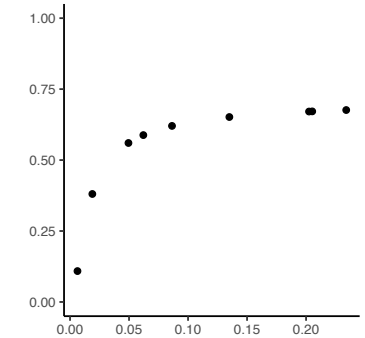
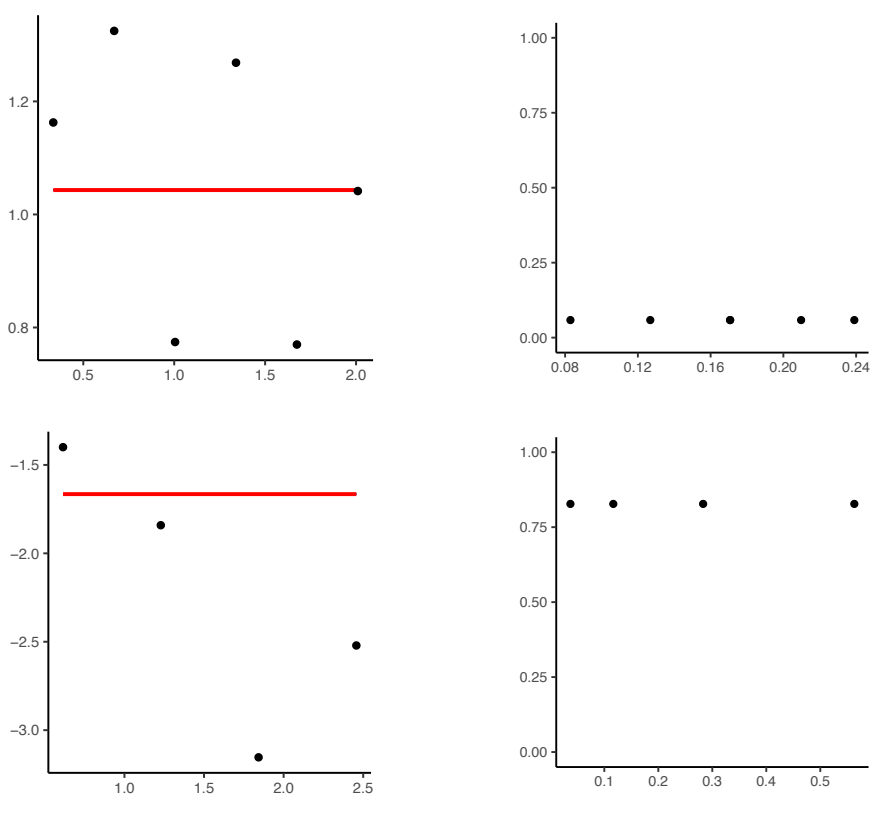
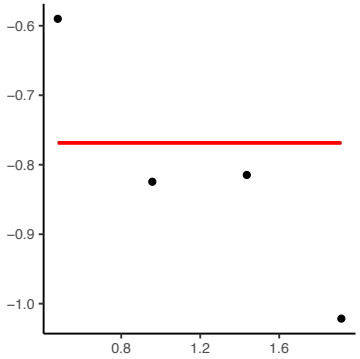
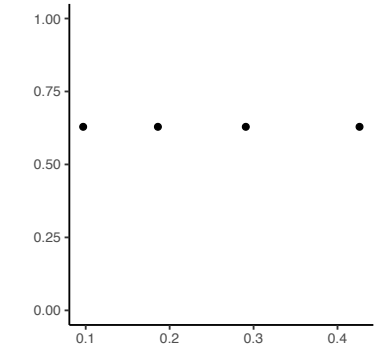
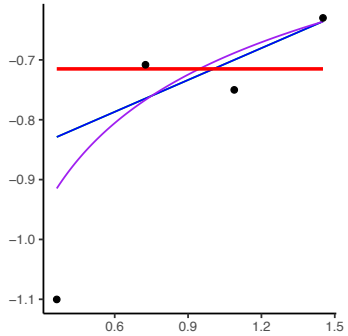
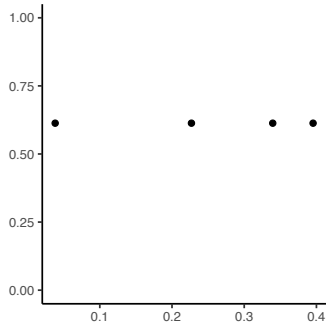
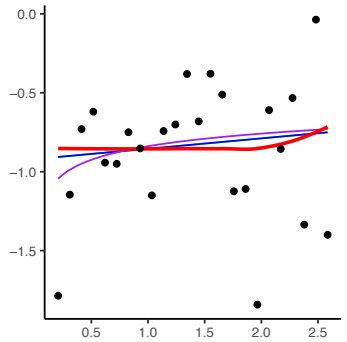
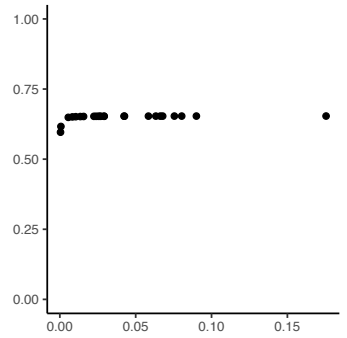
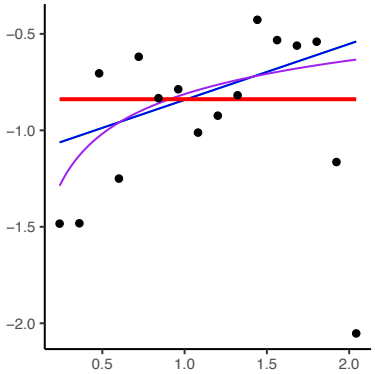
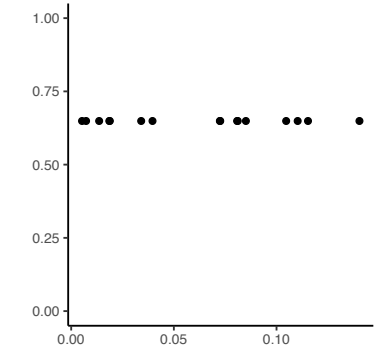
		4		
Elephas maximus	Robinson, 2012	1		

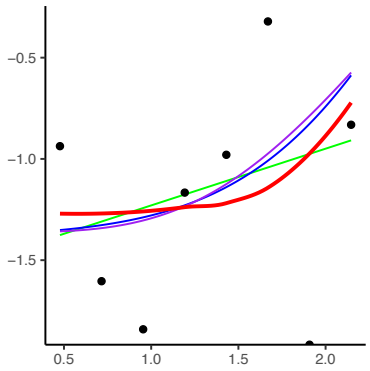
Falco columbarius	Espie, 2000	1
Ficedula albicollis	Gustafsson, 1990	1

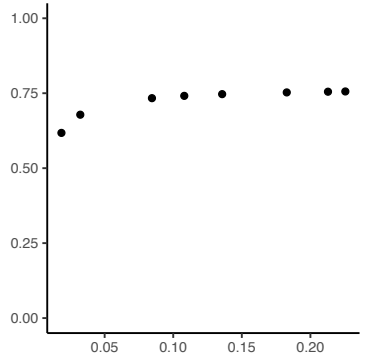


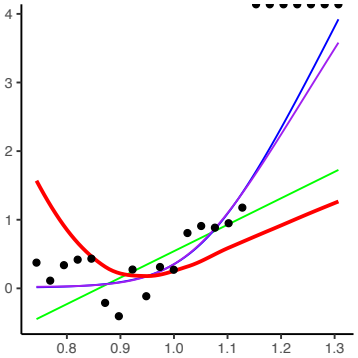
Ficedula hypoleuca	Potti, 2013	1		
		2		

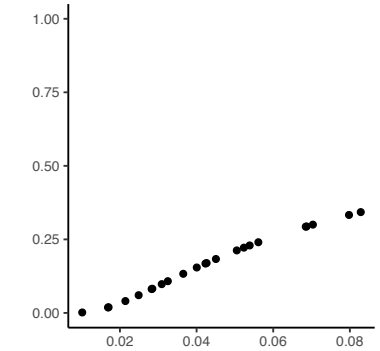
Galba cubensis	Gutierrez, 2000	1		
		2		

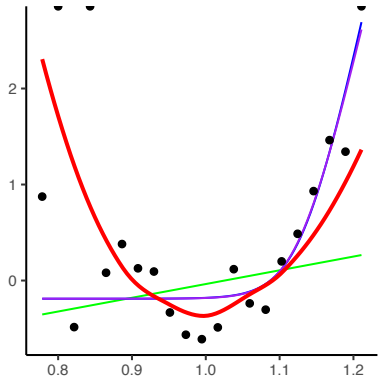
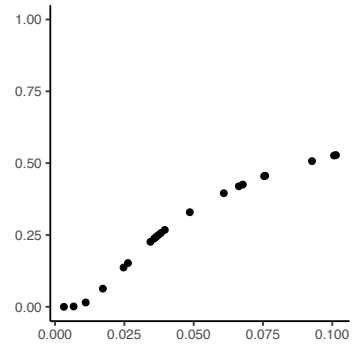
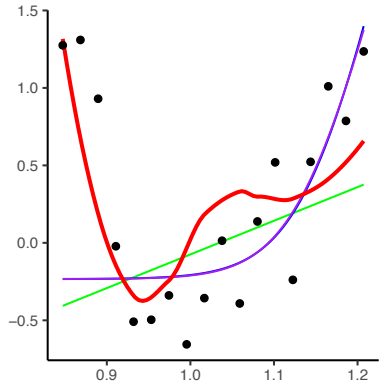
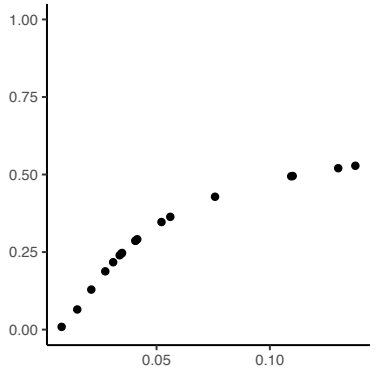
Gorilla beringei	Robbins, 2006	1
Helicoverpa armigera	Jha, 2012	1



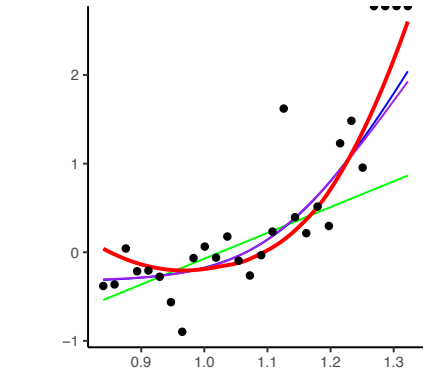


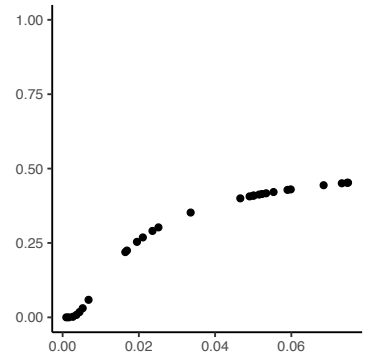


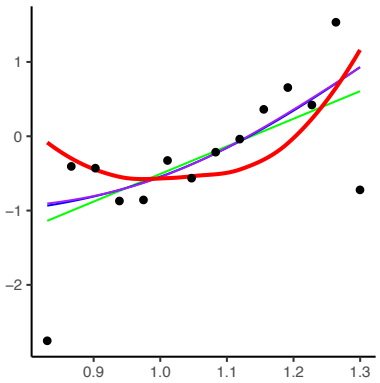


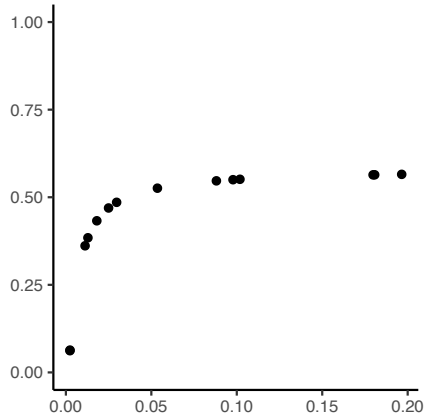
	Jha, 2012	1	 2 1 0 0.8 0.9 1.0 1.1 1.2	 1.00 0.75 0.50 0.25 0.00 0.000 0.025 0.050 0.075 0.100
		2	 1.5 1.0 0.5 0.0 −0.5 0.9 1.0 1.1 1.2	 1.00 0.75 0.50 0.25 0.00 0.05 0.10

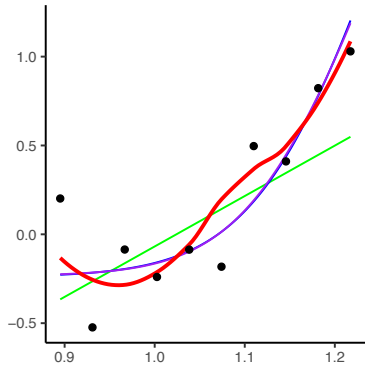
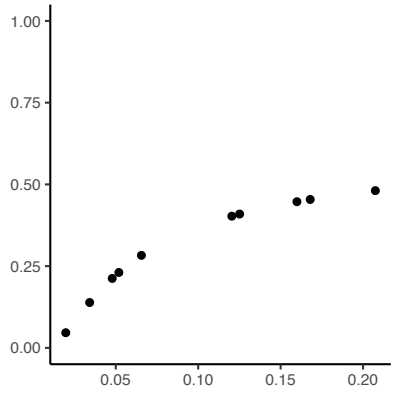
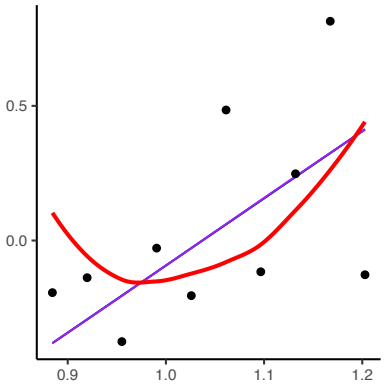
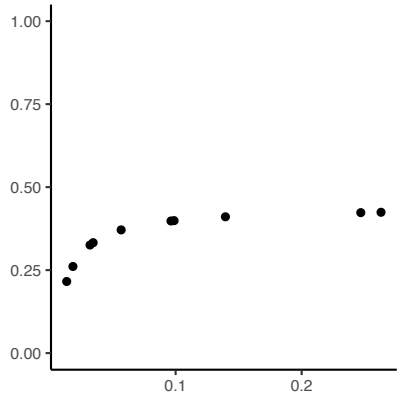
	Jha, 2014	1
	Liu, 2017	1

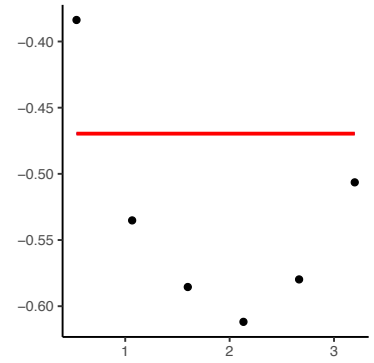
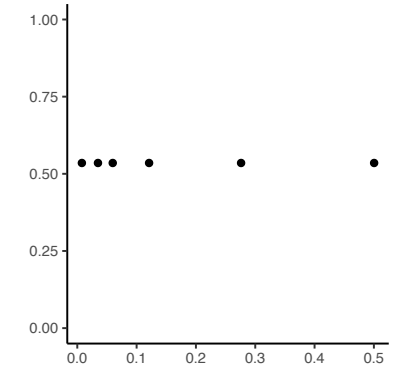
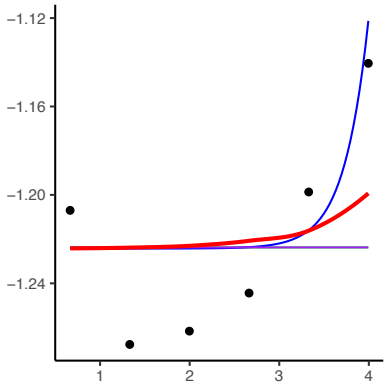
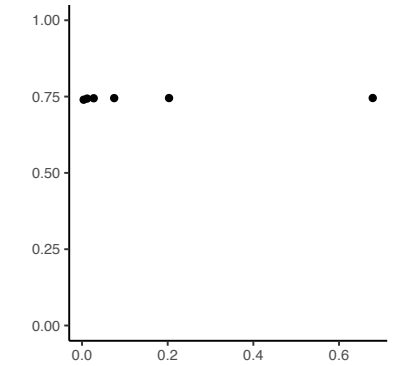








		2	 1.0 0.5 0.0 −0.5 0.9 1.0 1.1 1.2	 1.00 0.75 0.50 0.25 0.00 0.05 0.10 0.15 0.20
		3	 0.5 0.0 0.9 1.0 1.1 1.2	 1.00 0.75 0.50 0.25 0.00 0.1 0.2

Hirundo rustica	Balbontin, 2012	1		
		2		

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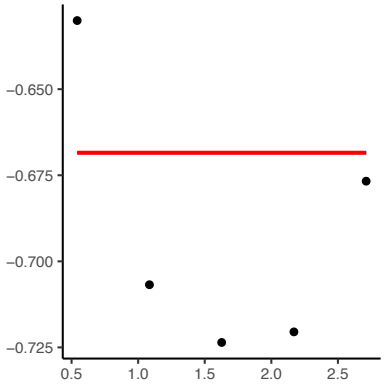
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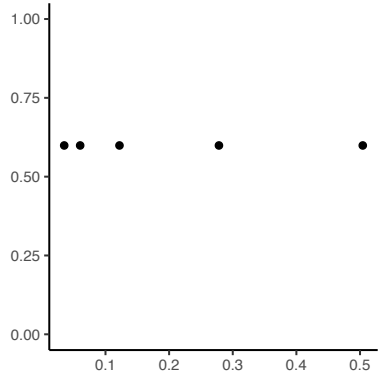
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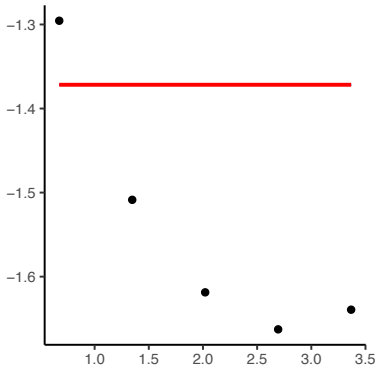
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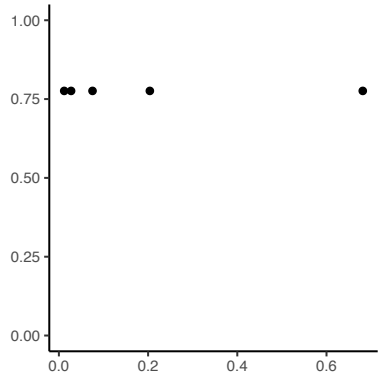
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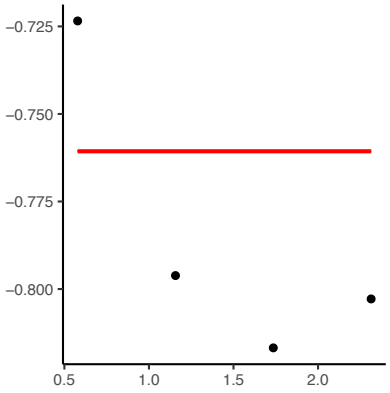
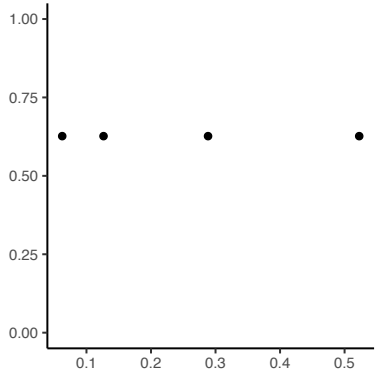
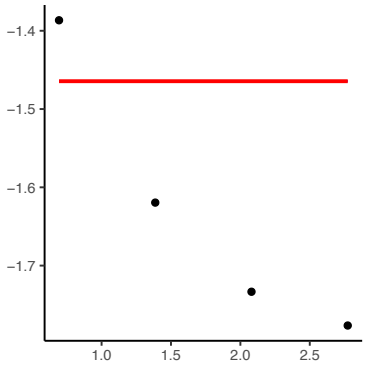
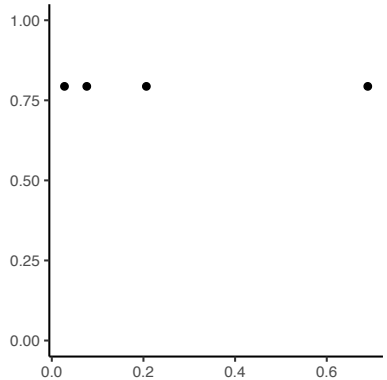
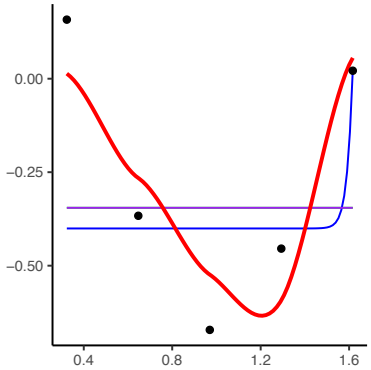
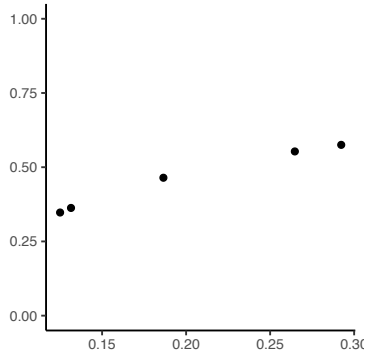
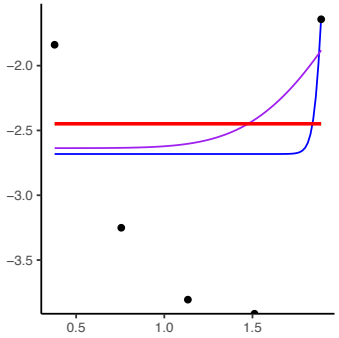
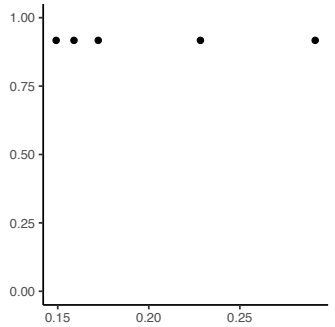
		5	 −0.725 −0.750 −0.775 −0.800 0.5 1.0 1.5 2.0	 1.00 0.75 0.50 0.25 0.00 0.1 0.2 0.3 0.4 0.5
		6	 −1.4 −1.5 −1.6 −1.7 1.0 1.5 2.0 2.5	 1.00 0.75 0.50 0.25 0.00 0.0 0.2 0.4 0.6

Lacerta vivipara	Richard, 2005	1
Lagopus muta japonica	Suzuki, 2013	1

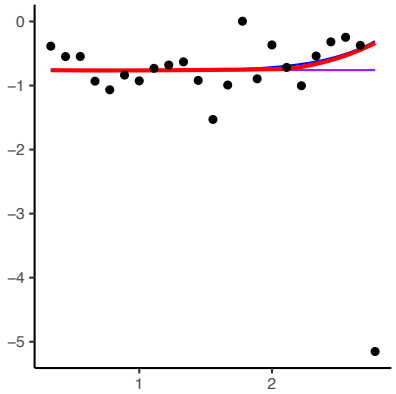


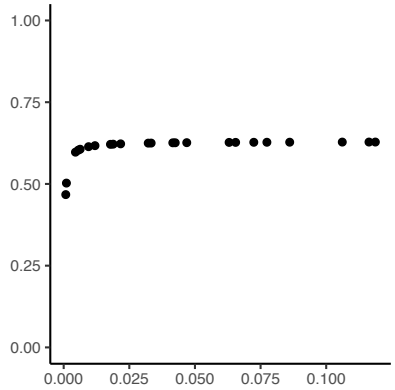


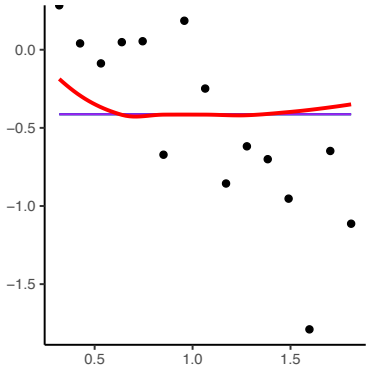


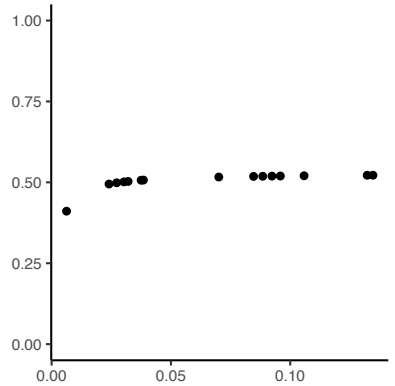


Larus audouinii	Oro, 2014	1
Larus californicus	Pugesek, 1983	1

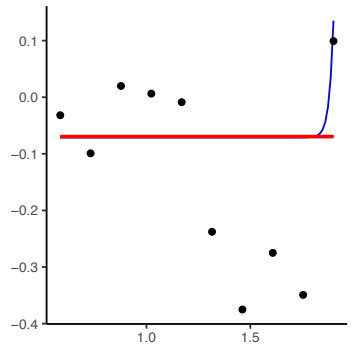


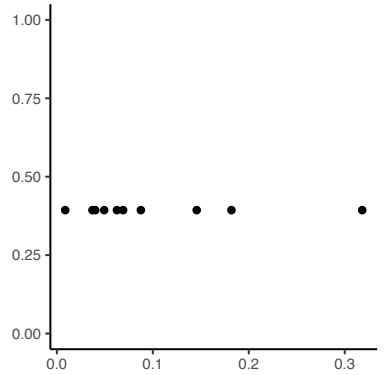


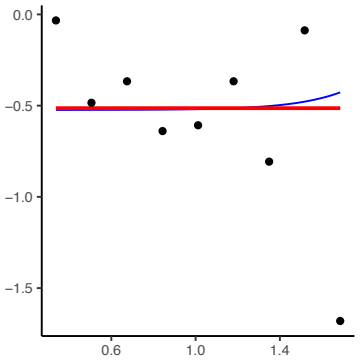


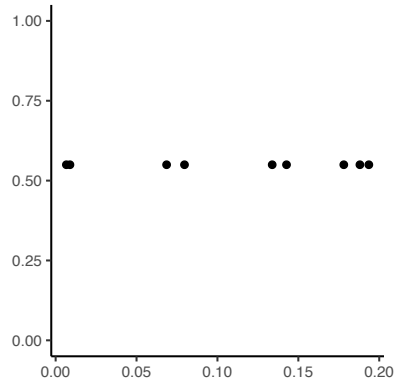


Larus heermanni	Vieyra, 2009	1
Larus novaehollandiae scopulinus	Mills, 1973	1

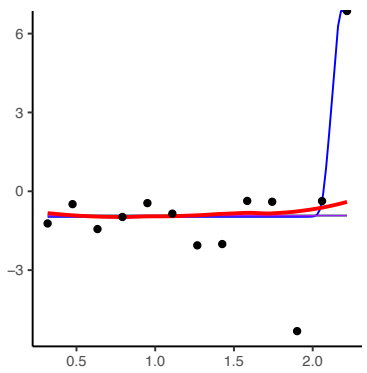


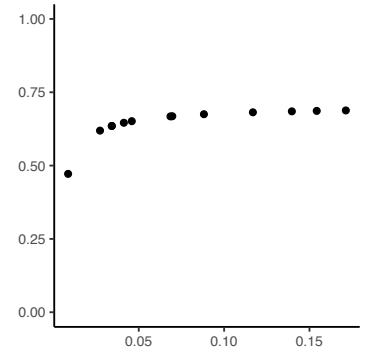


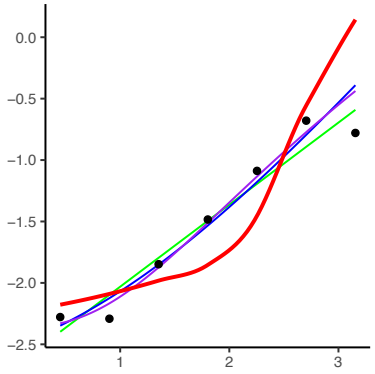


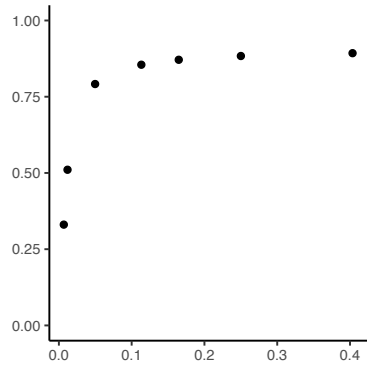


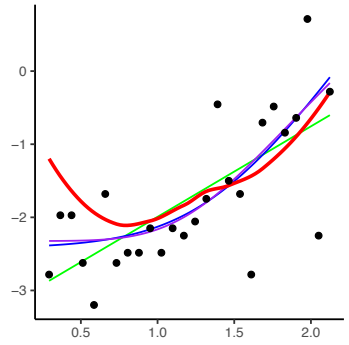
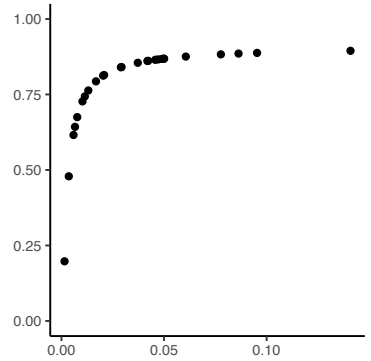
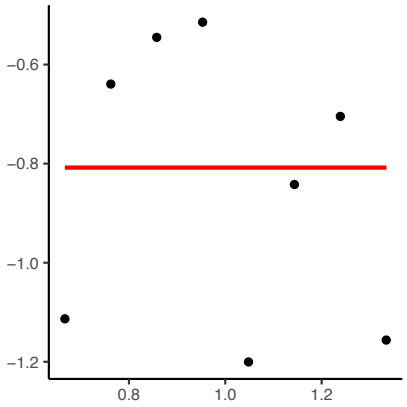
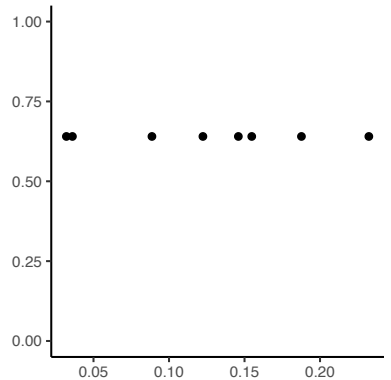
Lemur catta	Parga, 2005	1
Lobesia botrana	Moreau, 2016	1

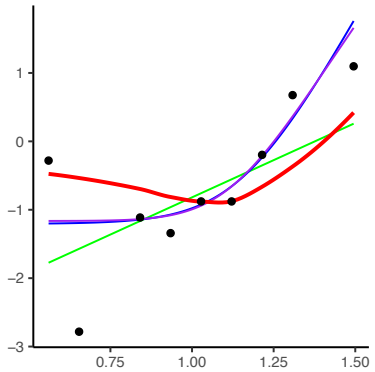
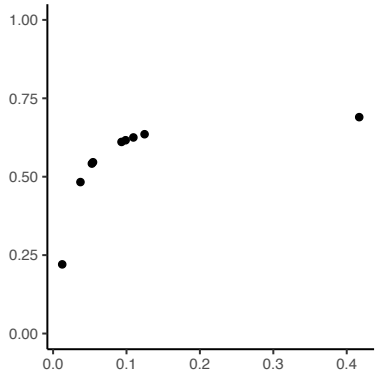
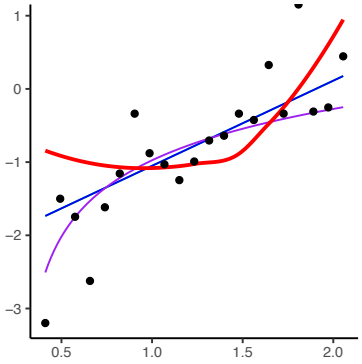
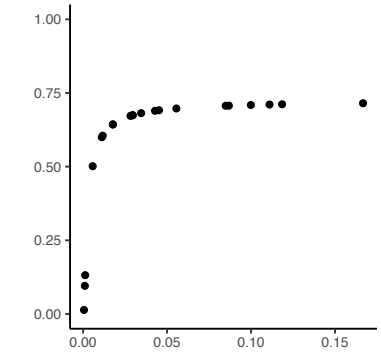




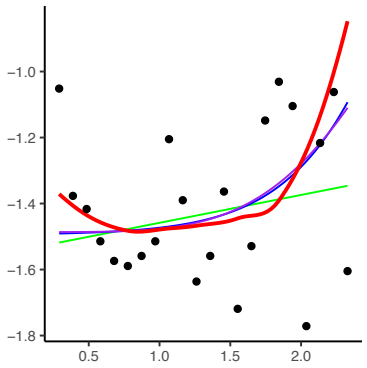


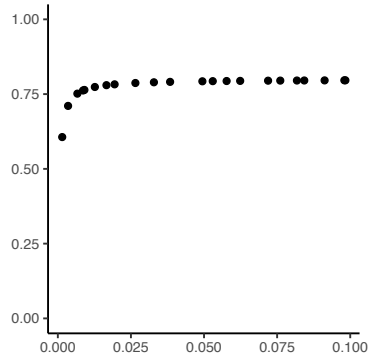


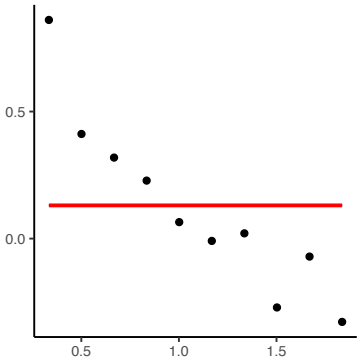
Lucilia cuprina	Readshaw, 1983	1		
		2		

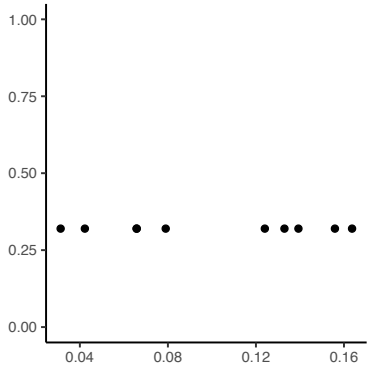
		3		
		4		

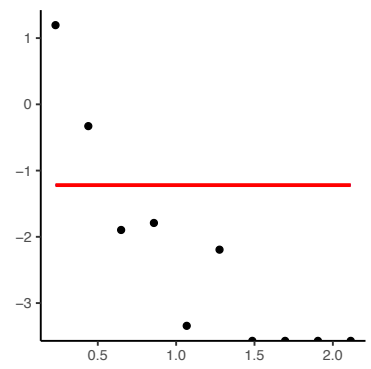
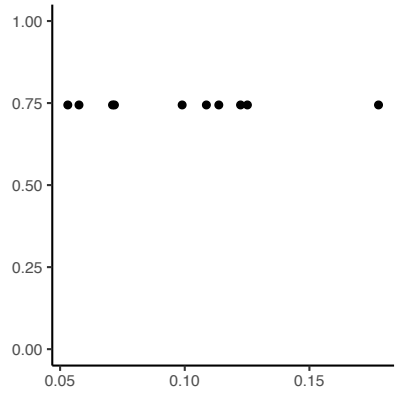
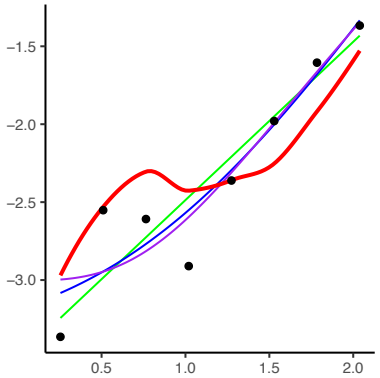
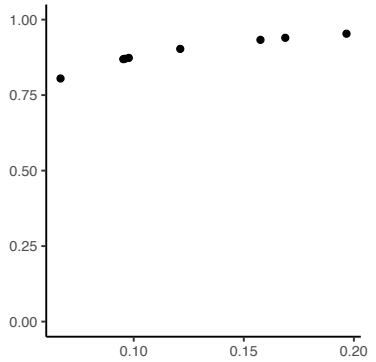
Macaca mulatta	Gagliardi, 2007	1
Milvus migrans	Blas, 2009	1

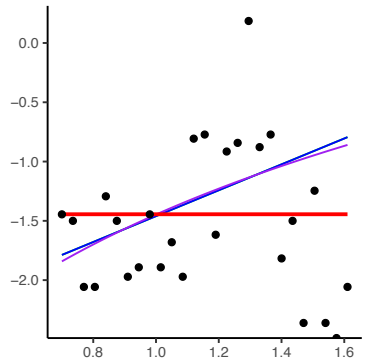
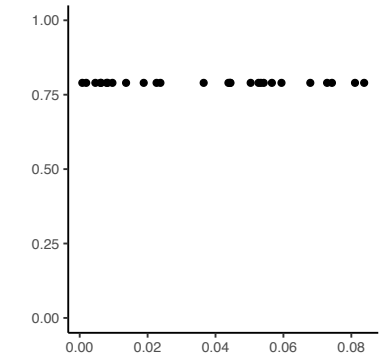
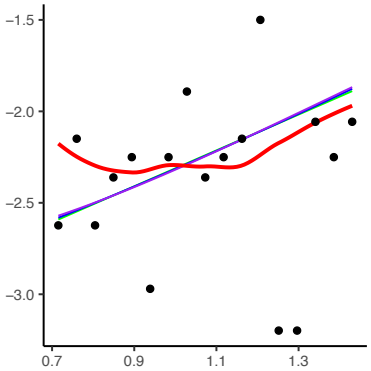
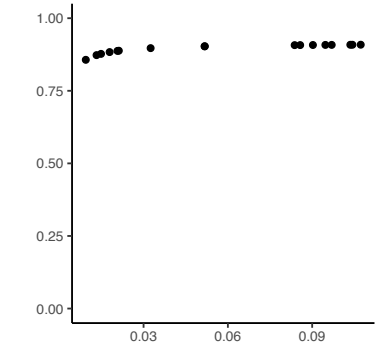








Nezara viridula	Kiritani, 1963	1		
	Kiritani, 1967	1		

Orius niger	Baniameri, 2005	1		
		2		

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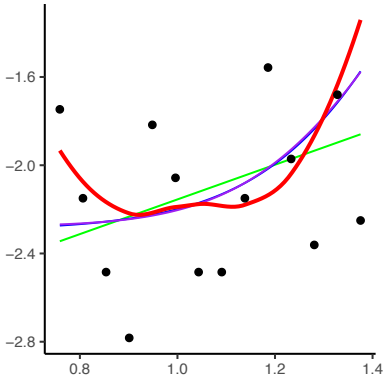
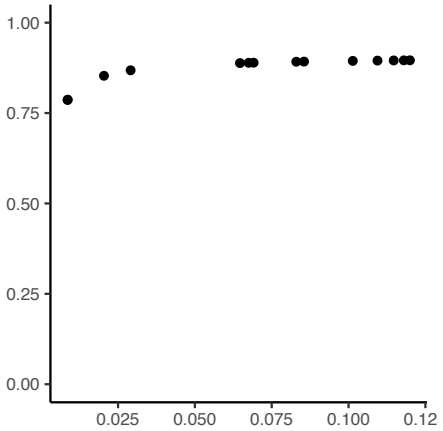
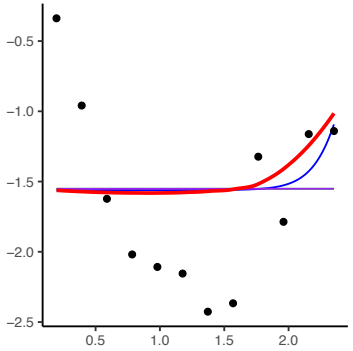
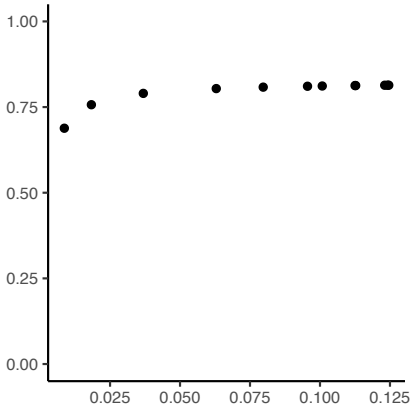
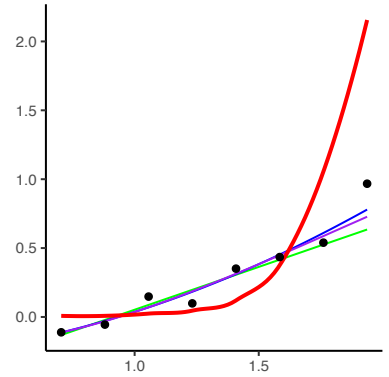
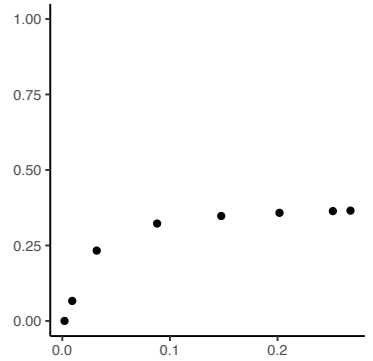
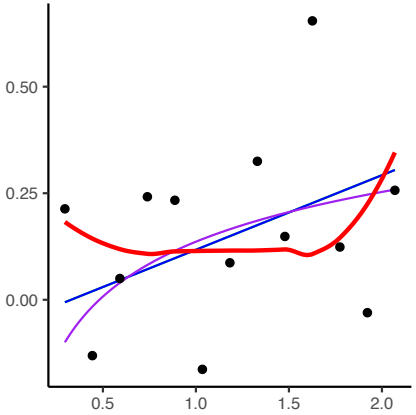
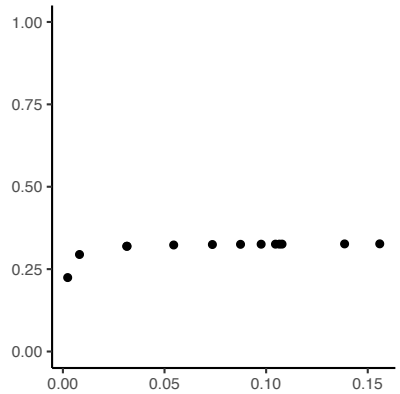
Ovis aries	Hayward, 2013	1



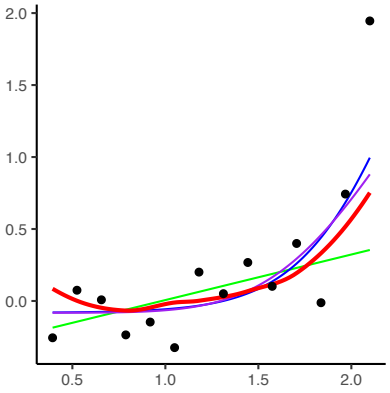


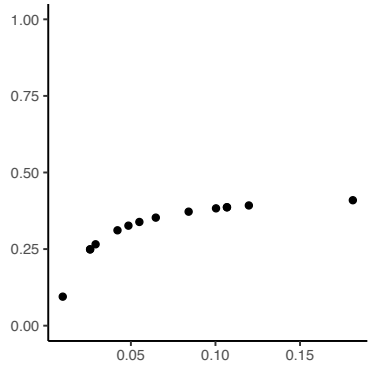


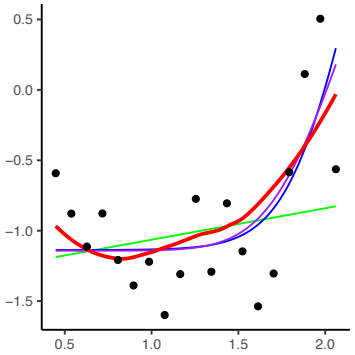


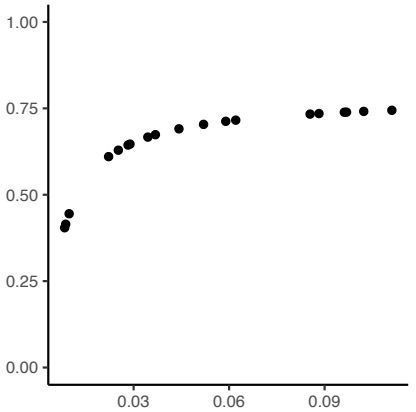
	Hayward, 2015	1		
Panthera leo	Packer, 1998	1		

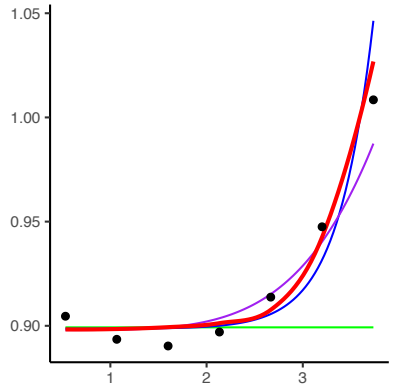
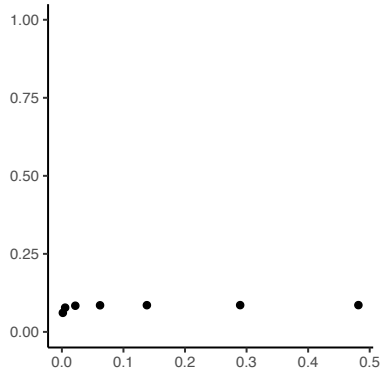
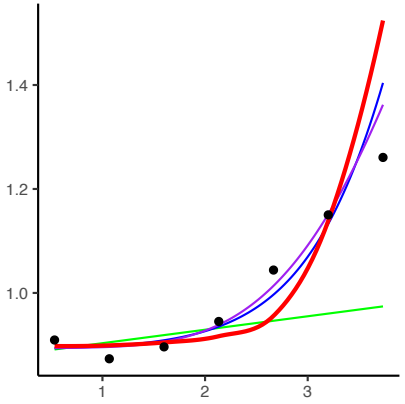
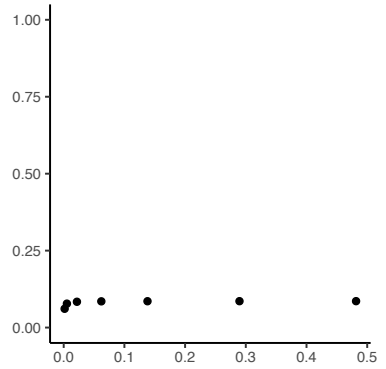
Panthera pardus	Balme, 2013	1
Papio anubis	Packer, 1998	1

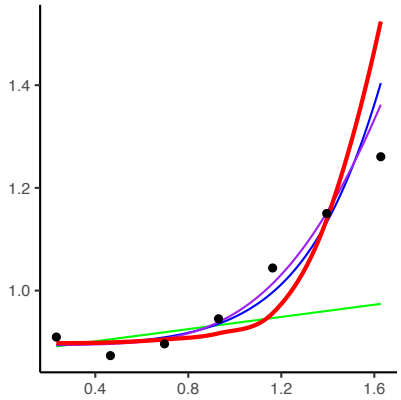
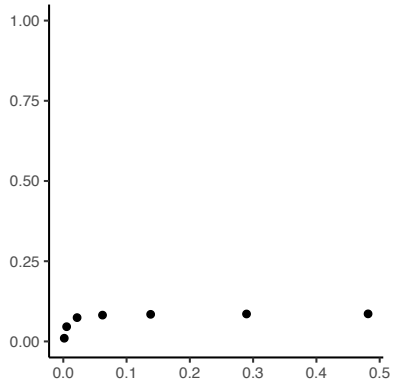
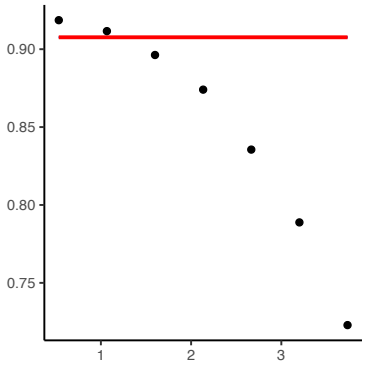
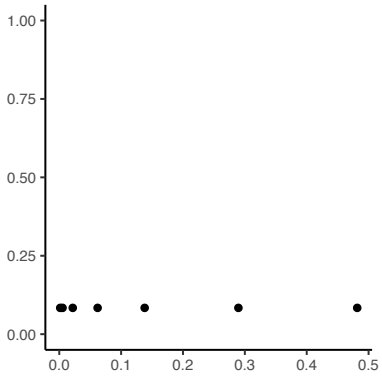








Parus major	Bouwhuis, 2009	1		
	Bouwhuis, 2010	1		

		2	 1.4 1.2 1.0 0.4 0.8 1.2 1.6	 1.00 0.75 0.50 0.25 0.00 0.0 0.1 0.2 0.3 0.4 0.5
		3	 0.90 0.85 0.80 0.75 1 2 3	 1.00 0.75 0.50 0.25 0.00 0.0 0.1 0.2 0.3 0.4 0.5

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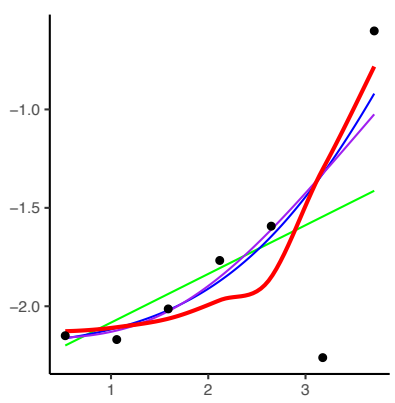
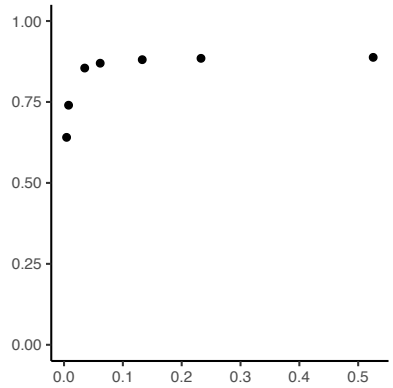
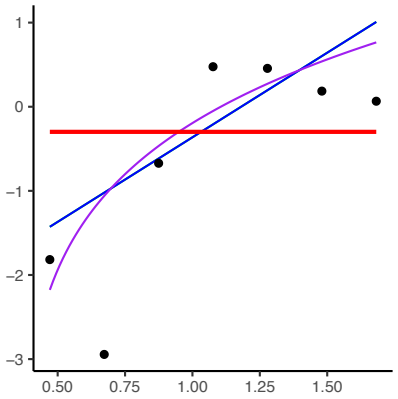
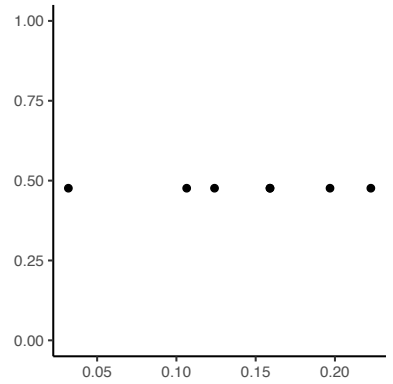
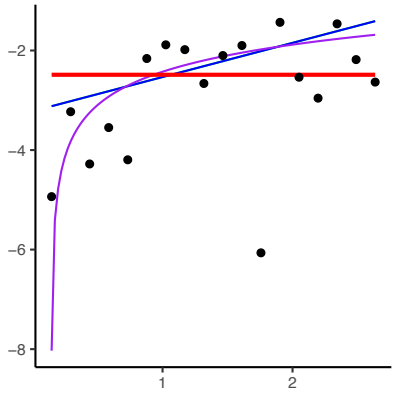
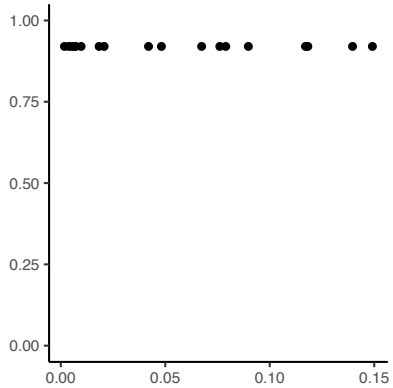
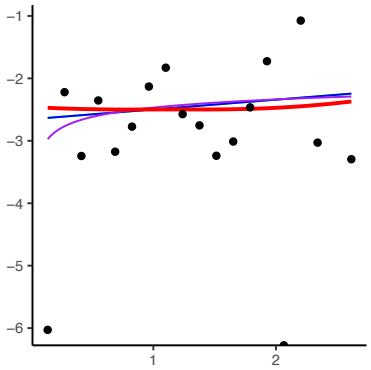
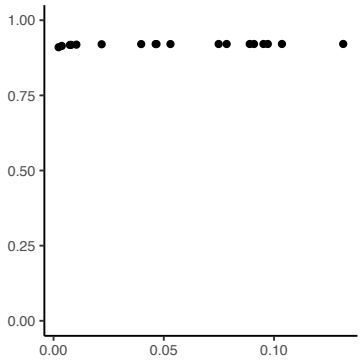
Physa acuta	Auld, 2014	1









Podisus nigrispinus	DeCastro, 2015	1		
		2		

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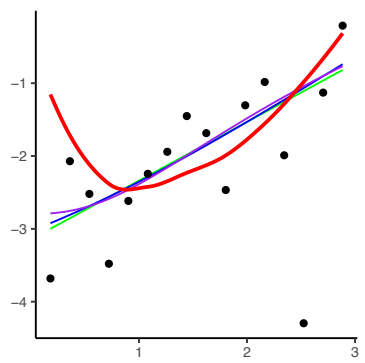
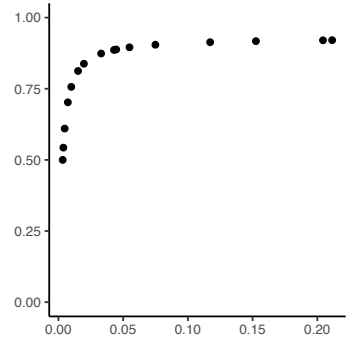
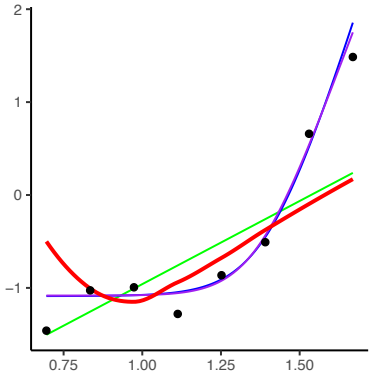
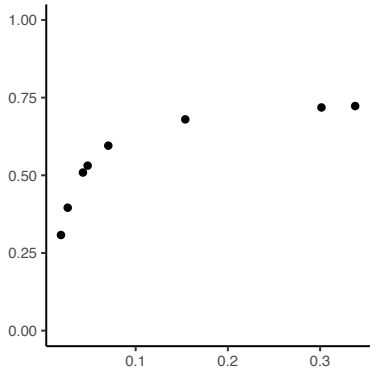
	Medeiros, 2000	1

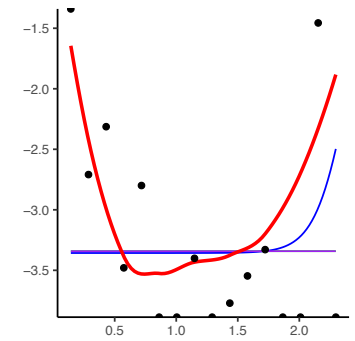


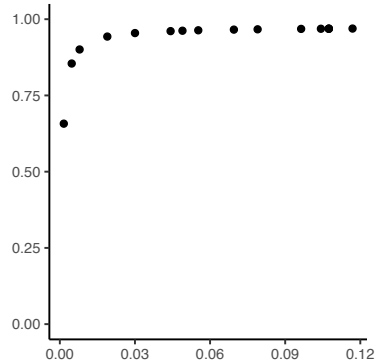


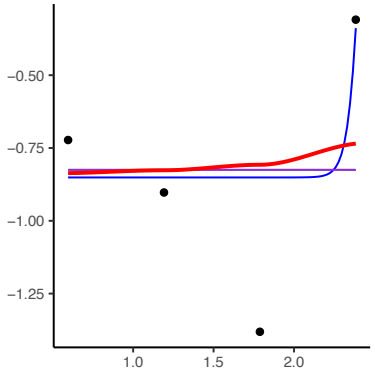


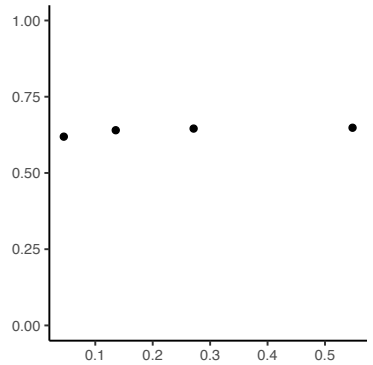


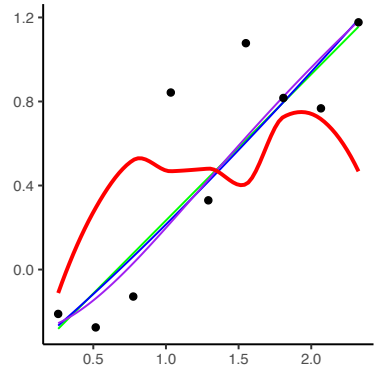
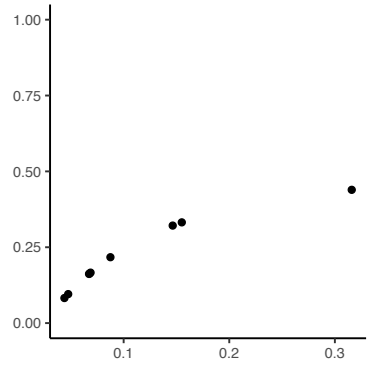
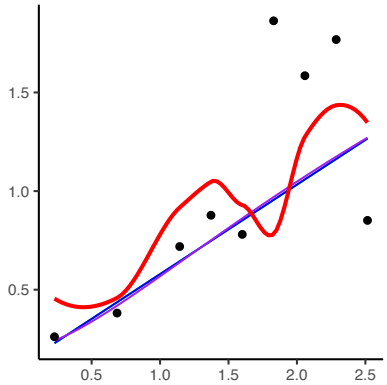
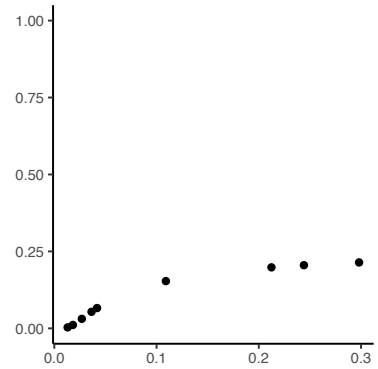
Rangifer tarandus	Jorgensen, 2015	1
Sialia mexicana	Keyser, 2004	1



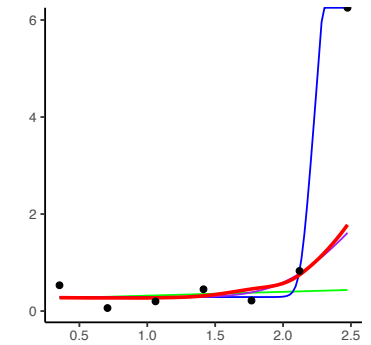


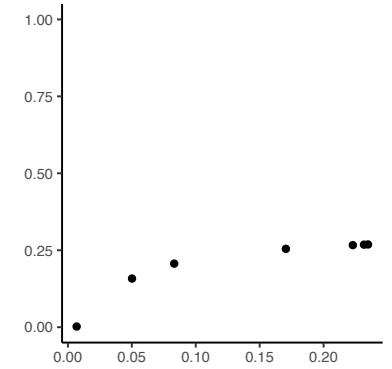


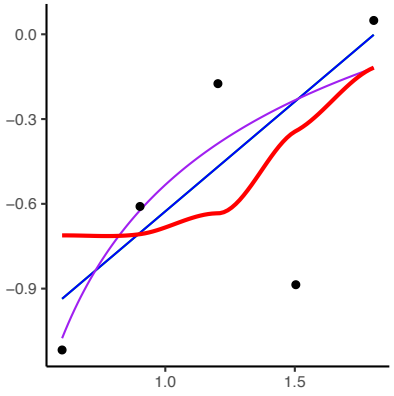


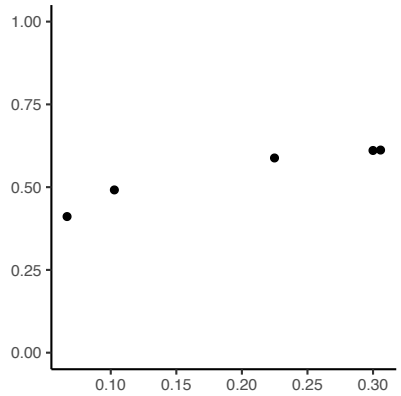
Spodoptera exigua	Rogers, 1996	1		
	Rogers, 1997	1		

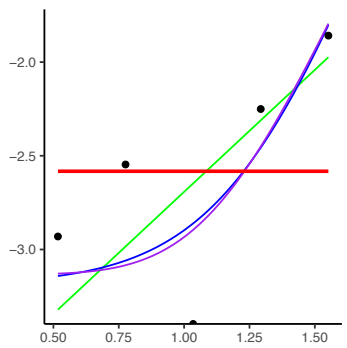
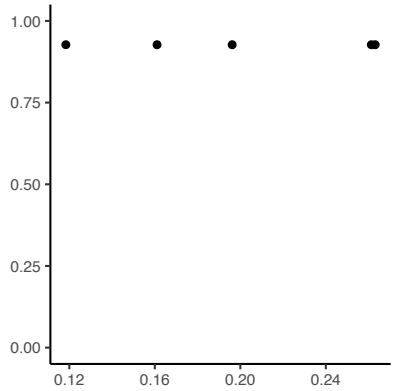
Tamiasciurus hudsonicus	Descamps, 2008	1
Tyrannus tyrannus	Murphy, 2004	1









Urocitellus columbianus	Skibiel, 2009	1		

Supplementary material from: "What can natural selection tell us about diversity of ageing rates across species?"

S4.1 Derivations for predicted vital rates

Age-specific mortality:

We assume that mutations act additively on the scale of mortality. From Charlesworth and Hughes (1996), we expect that at a mutation-selection balance, the amount of age-specific mortality should follow from the strength of selection for that mortality:

$$\mu(x) = \exp\left(-\frac{2\sum u}{\beta_{w,\mu(x)}}\right),$$

where $\sum u$ is the per-locus mutation rate summed over all loci. We assume that this is age-independent and sums to a non-negative constant *k*. Taking the natural logarithm of both sides,

$$ln\big(\mu(x)\big) = -\frac{k}{\beta_{w,\mu(x)}},$$

It follows that the natural logarithm of age-specific mortality should be inversely proportional to the negative selection gradient acting to favour mortality,

$$ln\big(\mu(x)\big) \sim -\frac{1}{\beta_{w,u(x)}} \qquad [1],$$

Age-specific fertility:

We assume that mutations act additively on the scale of fertility. From equation [11] in Charlesworth (2001), the loss of age-specific fertility caused by senescence at mutation-selection balance is,

$$m(0) - m(x) = k \exp(\mu x),$$

where $m(0)$ is the fertility at the youngest age class (assumed by the simple evolutionary theory to be maximized here). Re-arranged slightly, this is.

$$m(0) - m(x) = \frac{k}{\exp(-\mu x)}$$

However, this derivation makes clear from its context that the exponential term in the denominator is intended to be the strength of selection for fertility age at age *x*. In general, age-specific fertility selection is,

$$\beta_{w,m(x)} = l(x) \exp(-rx),$$

where *r* is the Malthusian growth rate and $l(x)$ is the cumulative rate of survival to age *x*, where $l(x) = \exp\left(\sum_1^x \mu(x)\right)$. In Charlesworth's application, *r* is set to zero (constant population size over time) and age-specific mortality is constant; this causes $\beta_{w,m(x)}$ to be $\exp(-\mu x)$. Substituting the more general expression describing age-specific fertility selection into Charlesworth's expression and re-arranging give us,

$$1 - \frac{m(x)}{m(0)} = \frac{k}{m(0)\beta_{w,m(x)}}$$

Given that *k* and $m(0)$ are constants, we expect that the proportion of fertility remaining at age *x* is inversely proportional to selection for fertility at that age.

$$1 - \frac{m(x)}{m(0)} \sim \frac{1}{\beta_{w,m(x)}} \qquad [2]$$